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THE EARLY DEVELOPMENT OF BEHAVIOR IN AMBLYSTOMA AND IN MAN*

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PHILADELPHIA

In *Amblystoma,* a genus of the amphibian *Urodela,* one finds an enlightening approach to the study of behavior. Unlike the *Anura* (frogs, toads, etc.), *Amblystoma* does not resorb the tail in metamorphosis from the tadpole or larval stage to the adult condition. The trunk and tail, on the other hand, continue throughout life as a flexible, integrated organ, the bending of which gives the animal its power of aquatic locomotion and, subsequently, becomes the primary component of locomotion on land. This lack of anatomic and physiologic specialization in the axial organization of *Amblystoma* makes the animal peculiarly adapted to the study of the development of behavior, and I have taken advantage of this adaptation by observing the behavior of different species of *Amblystoma* from the earliest movements till the general adult pattern of action is established.

As a result of this investigation of the development of behavior a general principle has been discovered which, in my opinion, has wide, if not universal, application in vertebrates. The principle may be stated thus: The behavior pattern from the beginning expands throughout the growing normal animal as a perfectly integrated unit, whereas partial patterns arise within the total pattern and, by a process of individuation, acquire secondarily varying degrees of independence. According to this principle, such an entity as a "simple reflex" never occurs in the life of the individual; complexity of behavior is not derived by progressive integration of more and more originally discrete units; the conception of chain reflexes as usually presented is not in accord with the actual working of the nervous system. On the other hand, within the total, ever expanding integrated organism as a whole, partial patterns emerge more or less and tend toward independence and dominance, but, under normal conditions, always remain under the supremacy of the individual as a whole. An inappropriate degree of independence or dominance of a partial pattern constitutes abnormality or perversion of behavior.

The evidence on which this interpretation is founded cannot be given in great detail here, but a statement of some of the more important items is essential to the purpose of this paper, which is to show that, so far as the early fetal movements of man are known, the same law prevails in the development of behavior in the human being as that which has been observed in *Amblystoma.*

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* From the Wistar Institute of Anatomy and Biology.

THE DEVELOPMENT OF BEHAVIOR IN AMBLYSTOMA

The earliest movements of *Amblystoma* are made by the most anterior part of the muscle system of the trunk.[1] They consist of a bending of that part of the trunk to one side or the other. As the animal grows older, this process of bending (flexure) extends farther and farther tailward until the whole segmental muscular system, trunk and tail, is involved. During the earlier period, all movements begin in the anterior region and progress tailward. This phase of development, therefore, consists of the expansion of a totally integrated pattern as a whole, and there are in it as yet no discretely individuated components.

These movements are at first relatively slow, but they increase in speed as the animal grows older, and, with this acceleration, the movements acquire locomotor value because each bend of the body progresses from the head tailward. In the earlier period, and still for a time after the whole trunk has become involved in the performance, the flexures are simple, that is to say, the body, in any particular act, bends only to the right or to the left. But as a result of still further increase in speed of action a second flexure in the opposite direction to that of the first in the anterior part of the body begins before the first flexure has passed tailward completely through the trunk. A series of such rapid flexures, alternating in first one and then the other direction, produces swimming. Locomotion in the water arises, therefore, by the expansion and speeding up of the original and primary pattern of behavior.

A merely casual observation of the earliest movements of *Amblystoma* in response to tactile stimulation would probably give the impression that they are simple reflexes of the conventional type; but there are several valid objections to such an interpretation. Both the afferent and the efferent paths consist of several or many neurons. Anatomically, therefore, the mechanism at the basis of these reactions differs materially from the conventionally adopted mechanism of the simple reflex. Furthermore, in their early stages of differentiation from neuroblasts and in their earliest functional period, the motor centers of the two sides constitute, in the region of the head, a single center that spreads across the floor plate or median line of the brain as a unitary structure,[2] so that half-centers, according to Graham Brown's [3] conception, do not exist as separate entities in *Amblystoma* of this early stage.

1. Coghill, G. E.: Anatomy and the Problem of Behaviour, London, Cambridge Univ. Press, 1929.

2. Coghill, G. E.: Correlated Anatomical and Physiological Studies of the Growth of the Nervous System in Amphibia: III. The Floor Plate of Amblystoma, J. Comp. Neurol. **37**:37, 1924.

3. Graham Brown, T.: On the Nature of the Fundamental Activity of the Nervous Centers; Together with an Analysis of the Conditioning of Rhythmic Activity in Progression, and a Theory of the Evolution of Function in the Nervous System, J. Physiol. **48**:18, 1914.

Physiologically considered, the flexure to the right, for example, might be regarded as antagonistic to flexure to the left, and on that basis the development of the swimming movement might be interpreted as a process of integration of primarily antagonistic reflexes, or the resolution of primarily discrete reflexes into chain reflexes. The latter interpretation seemed to me adequate at one time; but it has not yet been demonstrated that the sensory part of the conduction circuit is necessary for swimming in *Amblystoma;* whereas Tracy has shown that movements that are essentially like the swimming movement of *Amblystoma* occur in the toadfish before there is a functional exteroceptive or proprioceptive sensory field to participate in the reactions. In other words, movements such as constitute swimming in *Amblystoma* can be organized wholly within the motor mechanism of the nervous system. Furthermore, although the right and left flexures would appear to be potentially antagonistic to each other, I have found no evidence of actual antagonism between them in the normal development of swimming. As speed of movement accelerates, provision is made against such antagonism by the growth of collaterals from the motor neurons of each side into such relations with the motor neurons of the other as to produce inhibition on the side opposite a prevailing excitation. Here again, it should be emphasized that the mechanism is wholly on the motor side of the conduction circuit; the sensory system has no perceptible part in this inhibitory mechanism. But this growth of collaterals does not bring originally isolated motor centers into a new relation, for the motor mechanism of *Amblystoma,* as already mentioned, is originally unitary and median as regards the two halves of the body. These collaterals, developing simultaneously with the differentiation of the common unitary center into bilateral half centers, serve only to keep the whole motor mechanism in coordination. It has never been uncoordinated. On the whole, therefore, the evidence is against interpretation of swimming movements of *Amblystoma* as chain reflexes. It is, on the other hand, the expression of an expanding total pattern that has been perfectly integrated from the first.

When *Amblystoma* begins to swim, it has no motile appendages. As the external gills, fore limbs and hind limbs develop, their earliest movements are performed only as the trunk moves. The total behavior pattern, in other words, expands into the appendages as a primarily integrated system. This is true both for spontaneous movements and for movements in response to stimulation. This system of total integration dominates the appendages for a considerable time before they acquire any perceptible independence of action; that is to say, before local reflexes emerge from the total pattern by a process of individuation. And it is important to note that the early phases of this emergence of discrete limb movement are characterized by a gradual reduction of the

trunk component of the total pattern of trunk movement plus limb movement. The same principle holds also for the distal segments of the limbs: the earliest elbow or knee flexion occurs with movement of the limb as a whole.

Furthermore, the primary integration of the four limbs with the trunk dominates the early movements of walking. The primary component of walking is a slow swimming movement of the trunk and tail, which involves the limbs. The fore limbs are integrated with the movement of the trunk while the movement affects the anterior part, and as the trunk movement approaches the region of the hind limbs they also become involved. Accordingly, the hind limbs do not move synchronously with the fore limbs, but lag behind them by the interval required for the trunk flexure to pass from the region of the fore limb to that of the hind limb. The coordination of the limbs in the walking gait is therefore not a matter of nervous coordination directly between the limbs, but an integration of the limbs directly with the trunk. In its early phases of development, then, walking in *Amblystoma* is not a function of chain reflexes; it is a total pattern. Within this total pattern special adaptations in movement may eventually arise by the individuation of local reflexes. The antigravity component is probably of this nature.

Since, as shown elsewhere,[1] the capture and swallowing of food obviously arises according to the same mode of development as just outlined for locomotion, it may be said that the behavior of *Amblystoma*, so far as the form of the pattern is concerned, develops, not by the integration of simple reflexes into chains, but by the expansion of an integrated total pattern and an individuation of partial patterns of more or less local character which normally acquire a greater or less degree of independence as occasion requires and as is consistent with the integrity of the individual as a whole.

AVAILABLE EVIDENCE ON THE ORIGIN OF REFLEXES IN MAN

The movements of the human fetus in utero have attracted attention for many years, but direct observation which permitted accurate description of the form of movement of young fetuses has been for the most part casual and fragmentary, except the researches of Minkowski and of Bolaffio and Artom. The studies of Minkowski have been continued consistently through a number of years, and his observations are remarkable for their detail and scope; those of Bolaffio and Artom, while less extensive than Minkowski's, are recent, and add materially to the knowledge on the subject.

In order to present the available information on the chronologic order of the development of movements in the human fetuses of the first six months as briefly as is consistent with clearness, tables 1 and 2 have been constructed. Table 1 summarizes the protocols which

TABLE 1.—*Summary of Observations of Minkowski, Recorded in 1923*

	Second Month		Third and Fourth Months							
Serial number	35	37	29	44[a]	45	46	50	40	52	47
Anesthesia G(eneral) or L(ocal)	L & G	..	G	G	L	G	G	L	L	G
Total length (except no. 35) cm.	2.2	4	5.5	6	7.5	8.5	10	10.5	13	14
General	—	+	—	—	+	?	—	..	..	..
Arms and legs	—	..	—	—	..	?	—	+	+	+
Respiratory, convulsive	—	..	..	..	..	..	..	..	..	..
Legs, twitching	—	..	..	..	..	..	..	..	..	..
Legs and arms, choreiform	—	..	..	..	..	..	..	..	..	..
Fingers and toes	—	..	..	..	..	..	..	..	..	..
Short	—	—	—	+	..	+	+	..	+	..
Long	—	—	—	+	..	+	..	..	+	..
Plantar, foot dorsal	..	..	..	..	+	..	..	..	..	..
Plantar, foot plantar	..	..	..	..	..	+	..	..	..	..
Plantar, foot rotation	..	..	..	..	..	+	..	..	..	..
Plantar, leg extension	..	..	..	..	+	..	..	..	..	—
Plantar, toes, small, dorsal	..	..	..	..	—	?	..	+	+	—
Plantar, toes, small, plantar	..	..	..	..	—	?	+	..	..	—
Plantar, toe, great, dorsal	..	..	..	..	—	?	..	+	+	—
Plantar, toe, great, plantar	..	..	..	..	—	?	+	..	..	—
Plantar, toes, spreading	..	..	..	..	..	..	..	+	+	+
Plantar, leg flexion	..	..	..	..	..	..	..	..	+	—
Fingers, flexion	..	..	..	..	..	..	..	..	..	+
Plantar, great toe stronger than others	..	..	..	..	..	..	..	..	..	..
Plantar, great toe alone, dorsal	..	..	..	..	..	..	..	..	..	..
Plantar, great toe alone, plantar	..	..	..	..	..	..	..	..	..	..
Plantar, toes, gripping	..	..	..	..	..	..	..	..	..	..
Abdominal	..	..	..	..	..	..	..	..	..	..
Plantar, isolated Babinski	..	..	..	..	..	..	..	..	..	..
Plantar, great toe after others	..	..	..	..	..	..	..	..	..	..
Plantar, toes contralateral, dorsal	..	..	..	..	..	..	..	..	..	..
Leg flexion and contralateral extension	..	..	..	..	..	..	..	..	..	..
Arms, withdrawn	..	..	..	..	..	..	..	..	..	..
Legs, lashing	..	..	..	..	..	..	..	..	..	..
Movements in both legs	..	..	..	..	..	..	..	..	..	..

	Four to Six Months																										
Serial number	30	38	34	+4′	23	+5′	+2′	32	33	25	48	+1′	+1′	+1′	44	+3′	26	24	+3′	+4′	41	(R) +5′	(L) +5′	+1′	25	+12′	49
Anesthesia G(eneral) or L(ocal)	L	G	..	..	L	..	..	L	L	L	G	..	..	..	G	..	G	L	..	..	..	..	..	..	L	..	L
Total length (except no. 35) cm.	16	16	17.5	..	18	..	..	20	20	21.5	22	..	..	..	24	..	27	28	..	..	28	..	..	..	32	..	32
General	..	—	—	..	+	..	..	..	..	..	..	..	..	..	—	..	..	..	..	..	..	..	..	..	..	..	..
Arms and legs	+	—	—	..	+	..	..	+	+	+	..	..	..	..	—	..	..	+	..	..	..	..	..	..	+	..	+
Respiratory, convulsive	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Legs, twitching	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..
Legs and arms, choreiform	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..
Fingers and toes	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..
Short	..	+	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	+	..	..
Long	..	—	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	+	..	..
Plantar, foot dorsal	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, foot plantar	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, foot rotation	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, leg extension	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, toes, small, dorsal	+	..	+	..	..	..	..	+	+	+	..	..	..	..	..	..	..	..	..	..	+	..	..	..	+	..	+
Plantar, toes, small, plantar	..	+	..	+	..	+	+	..	..	—	+	+	+	+	..	+	+	..	..	+	..	+	..	+	..	+	..
Plantar, toe, great, dorsal	+	—	+	—	+	+	..	+	+	+	..	..	..	..	..	..	..	+	..	..	+	..	+	..	+	..	+
Plantar, toe, great, plantar	..	—	..	—	..	..	+	..	..	—	—	+	..	+	..	—	+	..	..	—	..	+	..	—	..	..	..
Plantar, toes, spreading	+	..	+	..	+	..	..	+	+	+	..	..	..	..	..	..	..	+	..	..	..	..	+	..	+	..	+
Plantar, leg flexion	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	+	..	..	..	+	..	+
Fingers, flexion	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, great toe stronger than others	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, great toe alone, dorsal	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, great toe alone, plantar	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, toes, gripping	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Abdominal	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, isolated Babinski	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	+	..	..	+	..	..	..	..	..	..
Plantar, great toe after others	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Plantar, toes contralateral, dorsal	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..
Leg flexion and contralateral extension	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..
Arms, withdrawn	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..
Legs, lashing	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..
Movements in both legs	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..

Minkowski [4] gives in detail in his contribution on the development of the plantar reflex; table 2 presents summaries of all the other explicit, individual records that I have been able to find on the movement of fetuses of similar ages. In his study of the plantar reflex, Minkowski reports on twenty-five fetuses varying from 2.2 cm. crown-rump length to 32 cm. total length. He expresses serious doubt, however, about one of these specimens (number 39, of 2 months) as to whether the movements could be regarded as true reflexes, for the reason that he used electrical stimulation in this case. I have, therefore, omitted the data for this fetus from the graphic record of table 1, thus reducing the number to twenty-four. In table 1 the upper horizontal line sets off, above, the descriptive matter relating to the fetuses from the kinds of spontaneous movements below; the second horizontal line marks off the spontaneous movements from those movements which were in response to stimulation, and which are, therefore, called reflexes. Numbers 35 and 37, included in the brackets, mark off the records of fetuses of the second month from those of the third and fourth months. The brackets including numbers 29 to 47 separate the records of the fetuses of the third and fourth months from the record of those of from 4 to 6 months. In the top row, reading to the right, are given the serial numbers which Minkowski attached to the individuals (there are two numbers 25, one of 21.5 cm., the other of 32 cm.). In the next horizontal row the method of anesthesia used in each case is indicated, whether general, "G," or local, "L." In the third horizontal row are indicated the lengths of the several fetuses, all referring to total length except the first, no. 35, which is crown-rump measurement. In the vertical columns assigned to the several specimens, as indicated at the top, are entered the reactions as indicated at the left of the table. A blank space signifies that Minkowski made no record concerning the particular reaction in the specimen indicated; a plus sign signifies that he recorded that the corresponding reaction occurred; a minus sign signifies that he recorded that such a reaction did not occur. For specimens 34, 23, 48, 44, 24, 41 and 25, Minkowski explicitly recorded more than one test. The time transpiring between the successive tests is indicated in minutes by the numbers following the serial numbers in each instance.

In table 2 there are represented records of twenty-seven fetuses, of the first 6 months, which are reported by various authors as follows: one each by Erbkam,[5] Krabbe,[6] Strassmann [7] and Zuntz,[8] two by Yanase,[9]

4. Minkowski, M.: Zur Entwicklungsgeschichte, Lokalisation und Klinik des Fussohlenreflexes, Schweiz. Arch. f. Neurol. u. Psychiat. **13**:475, 1923.

5. Erbkam: Lebhafte Bewegung eines viermonatlichen Foetus, Neue Ztschr. f. Geburtsk. **5**:324, 1837.

6. Krabbe, Knud: Les réflexes chez le foetus, Rev. neurol. **24**:434, 1912.

TABLE 2.—*Summary of Records of Fetuses Reported by Various Authors*

Observer	Y	S	M	B	Y	M†	M	B	B	M	M	M	M	M	M	Z	M	E	M	M	M	M	B	K	B	M	B
Length, crown-rump	2	2.2	3.5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Length, total	..	..	..	5.5	5.7	6.5	7	7	7	8.5	10	11	13	13.5	15	15	16	16+	19	20	21.5	23	23	24	28	30	31
Arm (right) spontaneous	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Arms and legs, spontaneous	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..
Intercostal muscles, spontaneous	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Temporal muscles, spontaneous	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Fingers, isolated spontaneous movement with trembling of thumb	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Mouth opened and closed, with arm movements, spontaneous	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	+	..	..	..	..	..	..	..	..
Great toe, dorsal, spontaneous	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..
Fingers, spontaneous	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..
Oral reflex, with leg movement, to touch on lip or tongue	..	..	+	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Orbicularis palpebrae reflex with action of both legs, to touch on eye	..	+	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Shoulders, arms, legs, symmetrical, to slight jar	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Arms and legs, reflex	..	..	..	..	+*	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	+	..	..	..	..
Thorax reflex, to touch with finger	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Grasping reflex, toes	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Patellar reflex, extension at knee	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Patellar reflex with action of opposite leg and both arms	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Arm adduction and rotation to touch on palm	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Thigh flexion to touch on leg	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Shoulder elevation repeatedly to pin prick in cerebral cortex	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Reflexes stronger after ablation of cerebral hemispheres	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..
Abdominal reflex, bilateral, to percussion	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	+	..	..	..	+	+	..	..	..	..
Arm flexion, adduction, rotation, leg movement on same and opposite sides, and head rotation, to mechanical stimulus on arm	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Head rotation, leg and both arms, to stimulus on arm	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Legs bilateral to stimulus on one	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Head, trunk, shoulders, arms, legs, simultaneously, to touch on body	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..
Chin elevation to touch on lower lip	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Anal reflex with strong movement of both legs	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Mouth movements, flexion both arms, head drawn backward, to touch on hand	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Head moved to one side	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..	..
Respiratory, mouth opening, head side to side, flexion of arms and legs	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..
Plantar reflex, foot and toes plantar without great toe	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..
Leg flexion, contralateral extension, to pinching	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..	..
Toes, extension and spreading, with leg flexion to touch	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..
M. orbicularis palpebrae reflex	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..
Mouth opened and closed, chin elevated, to touch on tongue	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..	..
Extension of thorax with retraction of head backward	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..	..	..
Toes, plantar flexion without great toe	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	+	..	..	..	..
Arm adduction, to pin scratch in pectoral region	..	.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..
Respiration, patellar reflex, bilateral, achilles reflex	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	+	..
Deep breathing movements	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+	..	..

* Electrical stimulation.

† It is not certain that all the records in this column are for one individual.

six by Bolaffio and Artom,[10] and fifteen by Minkowski.[11] Only records that are explicitly for a particular individual are included. In the upper row reading to the right the letters identify the observer: Y, Yanase; S, Strassmann; M. Minkowski; B, Bolaffio and Artom; Z, Zuntz; E, Erbkam. In the second row from the top the crown-rump length of the fetus is given; in the third row, the total length. In the columns reading down from the successive records of length are recorded with a plus sign the movements of the respective fetuses according to the descriptions indicated in the left of the table. Of Minkowski's specimens included in table 2, those of 3.5, 6.5, 7, 11, 13.5, 15, 19, 23 and 30 cm. in length are obviously not identical with any of the specimens used in his study of the plantar reflex as summarized in table 1; but in that study Minkowski used specimens of 8.5, 10, 13, 16, 20 and 21.5 cm., and these may be the specimens of those respective lengths included in this group. The minimum number of fetuses, therefore, represented in these two tables is forty-five, with a possible maximum of fifty-one.

In another contribution, Minkowski[12] reports on sixteen fetuses ranging from 5 to 23 cm. in length. In this group are specimens of 5, 5.6, 6.7, 9.5 and 12.5 cm. in length that obviously are not included in

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7. Strassmann: Das Leben vor der Geburt, Samml. klin. Vortr., 1900-1903, Gynäk., no. 132, p. 947.

8. Zuntz, N.: Ueber die Respiration des Säugethier-Foetus, Pflüger's Arch. f. d. ges. Physiol. **14**:605, 605, 1877.

9. Yanase, J.: Beiträge zur Physiologie der peristaltischen Bewegungen des embryonalen Darmes, Arch. f. d. ges. Physiol. **117**:345, 1907.

10. Bolaffio and Artom: Ricerche sulla fisiologia del sistema nervoso del feto umano, Arch. di sc. biol. **5**:457, 1923-1924.

11. Minkowski, M.: Réflexes et mouvements de la tête, du tronc et des extrémités du foetus humain pendant la première moitié de la grossesse, Compt. rend. Soc. de biol. **83**:1202, 1920; Ueber Bewegungen und Reflexe des menschlichen Foetus während der ersten Hälfte seiner Entwicklung, Schweiz. Arch. f. Neurol. u. Psychiat. **8**:148, 1921; Sur les mouvements, les réflexes et les réactions musculaires du foetus humain de 2 à 5 mois et leurs rélations avec le système nerveux foetal, Rev. neurol. **37**:1235, 1921; Ueber frühzeitige Bewegungen, Reflexe und muskuläre Reaktionen beim menschlichen Foetus und ihre Beziehungen zum fötalen Nerven- und Muskelsystem, Schweiz. med. Wchnschr. **3**:721 and 751, 1922; Zum gegenwärtigen Stand der Lehre von den Reflexen in entwicklungsgeschichtlicher und anatomisch-physiologischer Beziehung, Schweiz. Arch. f. Neurol. u. Psychiat. **15**: 239, 1924; ibid. **16**:133 and 266, 1925; Sur les modalités et la localisation du réflexe plantaire au cours de son évolution du foetus à l'adulte, Cong. d. méd. aliénistes et neurol. de France, 1926, p. 301; Neurobiologischen Studien am menschlichen Foetus, Handb. biol. Arb., 1928, part 5, p. 511; Ueber die elektrische Erregbarkeit der foetalen Muskulatur, Schweiz. Arch. f. Neurol. u. Psychiat. **22**: 64, 1928.

12. Minkowski, M.: Sur les mouvements, les réflexes et les réactions musculaires du foetus humain de 2 à 5 mois et leurs rélations avec le système nerveux foetal, Rev. neurol. **37**:1105, 1921.

the records entered on the two tables. The data for these five individuals, however, cannot be included in the tables because Minkowski does not describe them individually, but discusses them as a group and gives only general conclusions concerning their behavior. Nevertheless, these cases have value and receive attention in this paper. The total number, therefore, of human fetuses under 6 months which are recorded in such a manner as to make them useful for this study stands at a minimum of fifty.

Beyond the age of 6 months, human fetuses have been studied extensively, particularly by Minkowski, Bersot and Bolaffio and Artom. The behavior of these older fetuses is important for the study of the development of reflexes, but it is to the younger stages that one must look for the mode of origin of reflex behavior.

SPONTANEOUS MOVEMENT

The earliest spontaneous movement that has been observed in man is recorded by Yanase for a fetus of crown-rump length of 2 cm. (table 2). In this case the right arm moved back and forth about six or seven times; but Yanase considers that in the considerable time that elapsed after the fetus was removed and before it came under his observation, it may have made movements "in toto." In a specimen of 2.2 cm., Strassmann observed repeated movements of the arms and legs, whereas Minkowski describes the earliest spontaneous movements (number 37, 4 cm. total length, table 1) as vermicular and involving the body, head, arms and legs.

In the fetus 2 cm. in length which he observed, Yanase says that the fingers were separated only at the tips. In the specimen of 8.5 cm. (table 2) Minkowski says there were isolated finger movements with trembling of the thumb. Presumably he means by "isolated" that the fingers moved independently of any movements in the arms or hands, that is to say, the fingers and thumbs all moved in a common performance. Minkowski reports spontaneous finger movements also in a specimen of 20 cm. (table 2), and in another of 25 cm. In the latter he saw also spontaneous movements of the toes.

Beginning with Minkowski's specimen of 4 cm., in which there was slow vermicular movement of the head, trunk, arms and legs, the behavior pattern as indicated by spontaneous movement obviously expands through the extremities, as it does in *Amblystoma*. But in the two earlier specimens of 2 cm. and 2.2 cm. only limb movements were observed. On this evidence alone, therefore, it might appear that the axial musculature has not the precedence to and dominance over limb musculature in the human fetus that it has in *Amblystoma*. But in the evaluation of this evidence, at least three considerations must be kept in

mind: (1) the massive nature of the head of the human fetus would offer relatively much greater resistance to the contractions of axial musculature in man than in *Amblystoma;* (2) there is the possibility of trunk movements being unobserved, as Yanase explicitly indicates; (3) there are only two cases on record in which the fetuses were younger than Minkowski's specimen of 4 cm. that exhibited vermicular movements of head, trunk, arms and legs.

REFLEXES

Among the earlier reflexes many have an extensive pattern: mouth movements with leg movements (3.5 cm.); simultaneous symmetrical movements of shoulder, arm, legs (5.5 cm.); arms and legs (5.7 cm.); grasping movement of toes (6.5 cm.); contraction of musculus orbicularis palpebrae (oculi) with action of both legs (6.5 cm.); patellar reflex with action of opposite leg and both arms (7 cm.); flexion, adduction and rotation of the arm with movement of the other arm and both legs and rotation of the head in response to mechanical stimulus on the arm (7 cm.). During the corresponding period, however, certain localized reflexes have been recorded: patellar reflex in extension at knee (6.5 cm.); leg flexion (7 cm.); thorax reflex (5.7 cm), and abdominal reflex (7 cm.). The interrelation of these phenomena requires special consideration.

The Oral Reflex.—The earliest oral reflex in man is recorded by Minkowski [13] for a fetus of 3.5 cm. (table 2). But the opening and closing of the mouth in this case did not occur as a discrete act; it was allied with leg movement; that is to say, the reflex pattern involved the neuromotor mechanism from the trigeminal nuclei to the distal spinal centers. On the other hand, the earliest opening and closing of the mouth as a pure local reflex, that is, without participation of other parts, is recorded explicitly first at 21.5 cm. (Minkowski,[12] table 2). So far as my evidence goes, therefore, the oral movements in response to touch on or near the mouth occur at first as a component of a much larger pattern of movement and only later appear as a discrete reflex.

Limb Reflexes.—Isolated extension of the knee occurs at 6.5 cm., but the first reflex movement of the leg occurs with action of the other leg, of both shoulders and of both arms at 5.5 cm., and in alliance with the arms only at 5.7 cm. (table 2). Minkowski [12] records the patellar reflex in alliance with action of the other leg and both arms at 7 cm., whereas he apparently observed a pure patellar reflex at 6.5 cm. Authors acknowledge, however, that the tendon reflexes are difficult to make sure of in the early stages of fetal life, and this possible slight precedence of isolated patellar to the general patellar reflex pattern should not receive great emphasis.

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13. Minkowski (footnote 11, eighth reference).

Discrete reflex of the arm is first recorded at 7 cm.,[10] whereas the arm participated in a reflex pattern which included the legs at 5.7 cm.,[9] and in a pattern that included the other arm, the shoulders and the legs at 5.5 cm.[10] This earliest discrete reflex movement of the arm at 7 cm. involved both adduction and rotation, and was therefore not a simple reflex; it was a relatively complex or extensive pattern as regards the arm itself.

But still, at 7 cm. the extensive reflex pattern, including arms, legs and head, amounting almost to a total reaction, can be elicited. This particular reaction, observed by Bolaffio and Artom, seems to me to be of great importance, for at 9 cm. the same authors observed movement of both arms in response to touch on one, and response of both legs to touch on one. Nevertheless, they observed a reflex movement of one leg only at 7 cm. There is evidently a tendency here for the total pattern of which the discrete reflex is an organic component to recur in greater or less degree after the discrete reflex has become individuated. This principle is observed particularly by Minkowski and Bersot[14] in their study of later fetal and postnatal reflexes.

On the basis of reflex movements alone, therefore, it appears that discrete movement of the arm or leg does not arise as such, but emerges from a general pattern that involves other appendages, and probably more or less of the axial system.

But it will be recalled that the earliest observed spontaneous movement (at 2 cm.) was movement of the one arm only, and this might be regarded as contradictory to the conclusion just stated. But the observer, as previously noted, considered that in this case more extensive movements may have occurred. Moreover, the capacity for movement is not as completely expressed spontaneously as it is under excitation, for Bersot explicitly states that reflexes are elicitable in human fetuses after spontaneous movements cease, and the same feature is obvious in Minkowski's records. Furthermore, at 2.2 cm., the arms and legs participated in spontaneous movement. The difference in length of 0.2 cm. between the two specimens under consideration is probably negligible as regards chronologic order, for it may easily be due to differences in methods of measurement or to the manipulation of the specimen. This early spontaneous movement of the arm alone, therefore, does not seem to me to be necessarily antagonistic to the conclusion I have drawn from reflex movements of the appendages, that discrete movement of a limb emerges from a larger pattern of integration.

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14. Bersot, H.: Variabilité et corrélations organiques. Nouvelle étude du réflexe plantaire, Schweiz. Arch. f. Neurol. u. Psychiat. **4**:277, 1919; ibid. **6**:37, 1920; Développement réactionnel et réflexe plantaire du bébé né avant terme à celui de deux ans, ibid. **7**:212, 1920; ibid. **8**:47, 1921.

The Plantar Reflex.—The earliest recorded form of reflex in response to stimulation of the sole of the foot occurred in a fetus of 7.5 cm. (table 1).[4] It consisted of extension of the leg and dorsal flexion of the foot without positive movement of the toes. In a specimen of 8.5 cm. (number 46, table 1) Minkowski says that response was plantar flexion with rotation of the foot and "spontaneous inversion" of the reflex type. Minkowski does not say here explicitly that the toes moved when the foot moved, but he does say that in response to tactile stimulation of the sole of the foot there occurred movement of the foot outward with finally a plantar flexion, and that there occurred a "spontaneous inversion" of the reflex type, whereas in the context he defines "spontaneous inversion" as applying explicitly to toe movements. It seems certain, therefore, that the earliest movement of the toes that Minkowski observed as occurring in response to stimulation of the sole of the foot in his study of the development of the plantar reflex occurred in the same reaction with movements of the foot. This was at 8.5 cm., whereas the earliest movement of the toes that he observed without movements of the foot or leg occurred at 10 cm. It appears, then, that one has a complete history of the individuation of the pattern of movement of the toes as a group out of the larger pattern of movement of the leg, foot and toes.

It is significant also that the earliest response of the foot to plantar stimulation (8.5 cm., table 1) is not a simple movement; it is flexion and rotation. Similarly, the earliest movement of the toes, recorded without mention of the foot or leg, is the grasping reflex (not to plantar stimulation), which is an action of all the toes together, a total reaction of the toes. This occurs at 6.5 cm. (Minkowski,[13] table 2). But it is not until the fetus reaches 10 cm. in length that the plantar reflexes involve the toes only, and in this case Minkowski says that the toe movements were weak. In the fetus of 10.5 cm. there occur dorsal flexion of all the toes and spreading of the toes, the foot and leg not being mentioned (Minkowski, table 1). This reaction, in my opinion, may be regarded as a total reaction of the toes before discrete reaction of any particular toe occurs. Accordingly, in his 13 cm. fetus, Minkowski records what, in my judgment, is a total reaction of leg and toes (dorsal flexion and spreading of all the toes and flexion of the leg), whereas in a 14 cm. stage he saw spreading of the toes explicitly without flexion of the toes or movements of the foot or leg. Here, according to my interpretation, spreading of the toes has become individuated as a local reflex within the total leg-foot-toe system as represented in the 13 cm. stage.

In specimen 30, of the 16 cm. stage, Minkowski caught a phase of the development of the plantar reflex which has especial importance: in a total foot-flexion, toe-flexion and toe-spreading reaction, the flexion

of the great toe was stronger than that of the other toes. In other words, discrete flexion of the great toe was here just beginning to emerge from the general or total system of toe movement. Accordingly, in the other 16 cm. specimen (38) plantar flexion of the small toes occurred without participation of the great toe. Presumably, this isolation occurred because of the degree of individuation that the great toe had acquired for dorsal flexion as seen in specimen 30, or, in other words, inhibition is an essential factor in the process of individuation of the reflex. This interpretation is corroborated by Minkowski's explicit statement that the plantar flexion of the toes in specimen 38 (16 cm.) was a weak movement. It is further corroborated by specimen 34, a 17.5 cm. fetus, which in the first reaction gave a total toe-dorsal-flexion and spreading reaction and in the second reaction gave plantar flexion of the small toes only.

In the 18 cm. fetus (23) complete individuation of the mechanism of the great toe is functionally perfect in the second response, which consists of dorsal flexion of the great toe with plantar flexion of the small toes; but in the third response, possibly due to fatigue, the great toe falls back into the total system of toe-plantar-flexion. In two 20 cm. and one 21.5 cm. specimens (32, 33, 25) the total toe pattern prevails in the form of dorsal flexion and spreading for the most part, but in one of the 20 cm. specimens (32), in some of its reactions, the great toe emerges from the total pattern in a discrete dorsal flexion.

For the 21.5 cm. fetus Minkowski records explicitly the first typical isolated Babinski reflex in his protocols, but in his discussion he refers to the 18 cm. fetus as giving the earliest form of the Babinski sign. According to either of these statements, this typical reflex, according to the analysis presented here, has clearly arisen by a process of individuation within a total leg-foot-toe response.

The plantar reflexes of fetuses of 22 to 32 cm., as recorded by Minkowski, show frequent reversion to the earlier types of response through which the Babinski reflex developed. This phenomenon has been noted previously in relation to the limb reflexes.

The Eyelid Reflex.—The earliest observed contraction of the musculus orbicularis palpebrae (oculi) occurred with action of both legs at 6.5 cm.,[13] whereas discrete reflex action of this muscle occurred first at 21.5 cm.,[12] both observations being by Minkowski (table 2).

The Anal Reflex.—The earliest response to stimulation of the anal mucous membrane involved strong actions of both legs with contraction of the musculus constrictor ani. This was at 13 cm. (table 2);[13] and no isolated anal reflex is recorded for fetuses under 7 months. It appears, therefore, that this reflex also makes its appearance as a component of a larger reflex pattern.

Respiratory Reflexes.—The earliest movements which were explicitly designated as respiratory by the observer (Erbkam,[5] 16 cm.) involved opening of the mouth, movements of the head from side to side and movements of the arms and legs. In a fetus of 21.5 cm. (table 1) Minkowski describes the respiratory movements as convulsive. These observations indicate that the earliest respiratory efforts are virtually total reactions of the fetus. Nevertheless, reflex movements of the thorax are recorded for a fetus of 5.7 cm. and the abdominal reflex for one of 7 cm. For the latter age, spontaneous contractions of the intercostal muscles are also recorded (Bolaffio and Artom); however, these movements may have been direct responses of the muscles that were involved, for the muscles lie immediately beneath the skin and the stimulus was percussion or stroking. Furthermore, it is not clear that the reflex of the thorax observed by Yanase in the fetus of 5.7 cm. was not a direct response of the muscle to electrical stimulation, for he used electrical stimulation on this specimen and electrolytes may have been carried from the solutions on the fingers or instruments. Also the abdominal reflex observed by Bolaffio and Artom is open to question, for these authors used percussion on the abdominal wall in this instance. Although the reaction was bilateral, the mechanical impact on one side may have extended through the aponeurosis to the muscle of the opposite side. This interpretation is corroborated by the fact that to percussion on one side of the pectoral region, where the mechanical construction of the body is more rigid and the muscle masses of the two sides are more widely separated than in the abdominal region, Bolaffio and Artom could elicit only homolateral responses of the pectoral muscles in this same specimen. Furthermore, in a decerebrated fetus of 7 cm. they observed that the thigh participated with the abdominal muscles in a reaction to a unilateral excitation over the ribs. This would indicate that there is very early alliance of the limbs with action of the abdominal muscles, and that in all probability the abdominal reflex arises by individuation out of a larger pattern of integration or that it does not arise as a simple reflex and later become integrated with other reflexes into the respiratory reflex.

Having given arm movement (and probably trunk movement) at 2 cm., integrated arm and leg movement at 2.2 cm., and mouth movement integrated with movements of legs and arms (and probably trunk, for Minkowski describes the movements of 4 cm. fetuses as vermicular, involving head, trunk and limbs) at 3.5 cm., it appears that this expanding pattern invades the intercostal and abdominal muscles by the time the fetus reaches 7 cm. in length, at which age Bolaffio and Artom saw spontaneous contraction of the intercostal muscles and reflex contraction of the abdominal muscles (providing their interpretation of the abdominal reflex at that age is valid), and that this total pattern

acquires the vigor of action observed by Erbkam at 16 cm.; whereas the definitive respiratory pattern becomes individuated from the total pattern at about the 28 cm. stage as observed by Bolaffio and Artom, or by the 30 cm. stage as observed by Minkowski.[13] At any rate, I fail to find in the records of the behavior of human fetuses any dependable evidence that the definitive respiratory movement is acquired by the integration of discrete simple reflexes or by the development of chain reflexes. On the other hand, the evidence seems clear to me that this movement is acquired through the expansion of the integrated total pattern of movement as a whole, and by individuation within that pattern of the particular integrated system that the integrity of the organism requires as development proceeds.

BERSOT'S INTERPRETATION OF THE PLANTAR REFLEX

It is held by Bersot[15] that the earliest response to stimulation of the sole of the foot involves the toes only and that it consists of ventral flexion of the proximal phalanges and dorsal flexion (extension) of the others. These toe movements, Bersot says elsewhere,[16] are slow and weak but clear, and involve especially the four small toes, while the great toe moves with difficulty and more often remains entirely immobile. The toes, he says, seem to stretch out; they become fatigued rapidly and cease to respond after six or eight reactions. Only later in development, according to Bersot, is the limb withdrawn from the stimulus on the sole, with extension of the opposite limb, and eventually, the reaction becoming more and more generalized, the entire organism participates in the motor reaction. Finally, that is, at from 2 to 6 months of postnatal life, the reaction again restricts itself to narrower limits, and acquires the pattern of the definitive plantar reflex.

According to Bersot's interpretation, then, the reflex, of which the plantar may be regarded as typical, involves, when it is first performed, only the local part that is stimulated; that is to say, it is a local reflex, pure and simple, and only in later development progressively diffuses or irradiates more and more extensively until practically the whole organism participates in the reaction; and, finally, this process is reversed, and there takes place a localization of the response to the extent that only the local stimulated part may react to the appropriate stimulus. This theory of the development of the reflex, as proposed by Bersot, is explicitly adopted also by Minkowski.[17]

It appears that the later phase of the development of the plantar reflex, the phase of concentration, according to Bersot and Minkowski,

15. Bersot (footnote 14, fourth reference).

16. Bersot (footnote 14, third reference).

17. Minkowski (footnote 11, third reference).

is in agreement with my observation of the process which I call individuation in the development of reflexes in *Amblystoma*. The interpretation, however, that the plantar reflex begins with movement of the toes only, appears not to be in harmony either with my observation on *Amblystoma*, or with certain observations of Minkowski on the human fetus.

Minkowski,[18] in describing the behavior of four fetuses of from 5 to 5.5 cm. total length, as already noted, says that they performed cutaneous and proprioceptive reflexes, short and long, homolateral and crossed. Most often, he says, it was not a question of isolated reflexes, but of general motor reactions that were variable. Excitation of the foot, for instance, could provoke, besides a reaction of the stimulated leg, motor reactions in the two arms, the head and the trunk. Minkowski does not say that this stimulus on the foot might be on the sole, but in another place [4] he demonstrates that the plantar reflex as late as the 22 cm. stage is provokable by stimulation on the dorsum as well as on the plantar surface of the foot.

According to this principle, which is emphasized by both Bersot and Minkowski, that in the early stages of the development of reflexes there is no well defined localized specificity of the receptor field for the excitation of a particular reaction, the extensive reactions which Minkowski describes as involving movement of the trunk, head, arms and legs in response to stimulation on the foot in fetuses of 5 cm. in length may well be regarded as related to the local reaction of the foot and toes in exactly the same manner as the total reaction of the trunk and limb of *Amblystoma* is related to the local reflex of the limb. Furthermore, Minkowski records observations on a fetus of 7.5 cm. in length (table 1) that moved the foot and leg in response to stimulation on the sole of the foot. In this case the locus of the stimulus was specific for the plantar reflex, and, although movements of the toes were not observed, this reaction may well be regarded as a precursor of the typical plantar reflex of the toes, that is to say, a phase of the individuation of the reflex out of the total pattern, which the more extensive reactions just mentioned for 5 cm. fetuses may be considered to represent. The validity of Bersot's interpretation of the mode of origin of the reflex depends on whether the early movements of the human fetus lack integration.

INTEGRATION OR DIFFUSION IN THE EARLY MOVEMENTS OF THE HUMAN FETUS

In his report on a group of fetuses ranging from 5 to 23 cm. in length, Minkowski [12] says that the head turned from side to side, was elevated and depressed; the trunk curved and straightened, and the

18. Minkowski (footnote 11, first reference).

extremities flexed, extended and rotated. Minkowski does not make it certain that all the components of the behavior pattern, or any particular number of them, were operative in any given performance except that in some cases several articulations were involved simultaneously. He describes these movements, however, as asymmetrical, arrhythmic, diffuse and incoordinate. Nevertheless, he says in the context that one of these reactions may apply to more than one limb simultaneously. This would seem to contradict his statement that the movements are incoordinated. In another contribution [19] he says that spontaneous movements of four fetuses of from 5 to 5.5 cm. in length involve rotation, raising and lowering of the head; flexion and extension of the trunk; rotation, abduction and adduction, flexion and extension at the elbow, shoulder, wrist and hip, and that movements at several articulations take place simultaneously. He says also that excitation of the foot provoked, besides reaction of the excited leg, motor reactions in the two arms, the head and the trunk.

Furthermore, Minkowski says elsewhere [19] that the crossed reflex shows the first elements of coordination of reflex components, that is to say, the reflex in which both limbs move in response to stimulation of one limb only. Since he observes this pattern of reaction, as just noted, in fetuses of from 5 to 5.5 cm., he therefore must regard that coordination of movement occurs at that early age.

That there is utter lack of coordination in these early movements of fetuses, or that the movements that are called diffuse by Minkowski and Bersot are simply an aggregation of physiologically disconnected elements does not seem possible when the evidence is assembled from all available sources.

Minkowski himself describes the anal reflex of a 13 cm. fetus as involving with contraction of the musculus constrictor ani strong simultaneous movements of both legs backward. This reaction, particularly the simultaneous reaction of the legs, certainly expresses coordination of movements, and this is far within the age limits of the group of fetuses the movements of which he says are incoordinated. He describes also leg movements that occur simultaneously with movements of the mouth in fetuses of 3.5 and 6.5 cm. In the same two specimens both legs participated in what Minkowski designates as the eyelid reflex. The grasping movement of the toes which he describes for a fetus of 6.5 cm. must also express coordination. In a fetus of 10 cm., Minkowski says definitely, the shoulders moved at the same time with flexure of the trunk, neck and movements of the limbs. The simultaneity of action in all these components of a given performance must mean coordination. Minkowski reports also that he has seen in a fetus of 13.5 cm. the

19. Minkowski (footnote 11, fifth reference).

touch of a hand induce, besides flexion of the two arms, repeated movement of the mouth and simultaneous drawing of the head backward. Again, the simultaneity of action, which is explicitly described, must mean coordination. It seems to me, therefore, that Minkowski's particular descriptions of individual cases contradict his general statement previously cited that the movements of human fetuses of lengths from 5 to 23 cm., or from the ages of 2 to 5 months, lack coordination.

Bearing on this question also is the observation of a fetus of 5.5 cm. by Bolaffio and Artom. In this case, the authors say that a tapping with the finger lightly on the table on which the specimen lay in a dish elicited repeatedly strong symmetrical movement that involved elevation of the shoulder, abduction of the arm and flexion of the thigh and leg. These movements, they say, could be elicited at will for about three minutes. According to the authors' description, this fetus of 5.5 cm. exhibited consistently a definite behavior pattern that must, it seems to me, be interpreted as expressing coordination. I am led, therefore, to the conclusion that the term "diffuse" as used by Minkowski and Bersot cannot mean lack of coordination or integration.

THE SIMPLE REFLEX IN PHYSIOLOGY

Sherrington's conception of the reflex as a nervous function is well known, but it should be stated here in order to make a comparison of the purely physiologic with the embryologic interpretation, as presented in this communication, definite and clear.

"The unit reaction in nervous integration," Sherrington[20] says, "is the reflex." The "simple reflex" is that grade of coordination which makes "an effector organ responsive to excitement of a receptor, all other parts of the organism being supposed indifferent to and indifferent for that reaction." "In this simple reflex," he adds "there is exhibited the first grade of coordination." Nevertheless, he says explicitly, "A simple reflex is probably a pure abstract conception, because all parts of the nervous system are connected together and no part of it is probably ever capable of reaction without affecting and being affected by various other parts, and it is a system certainly never absolutely at rest." But nowhere in the conventional physiology of reflexes do I find the unity of the reflex with the total behavior pattern revealed in such clearness as I see it in the development of reflexes in *Amblystoma*. That these reflexes are in the same category with those of neurophysiology I have explained elsewhere.[1] In development, the "simple reflex" proves to be what Sherrington suspected it to be, "a pure abstract conception;" and while Sherrington regards it as necessarily affected by and affect-

20. Sherrington, C. S.: Integrative Action of the Nervous System, New Haven, Yale University Press, 1911.

ing "various other parts" of the nervous system, in development it appears as inextricably fused with the total behavior pattern. This feature of it I have treated more at length in my lectures on "Anatomy and the Problem of Behaviour." There I have shown how in its very beginning the exteroceptive reflex is conditioned by proprioceptive or postural conditions of the animal as a whole. From this total pattern the reflex emerges more or less in discreteness, but in the light of the knowledge of its development, to hold that it ever acquires independence from the total pattern so as to constitute a "unit reaction" is begging the question; and to found the physiology of the nervous system on an erroneous abstract conception is, to say the least, not working along the most direct line of progress. Neurophysiologists obviously appreciate that the "simple reflex" is not actually a simple affair, but unfortunately it is accepted by many writers on psychology as a real unit of structure and function, and larger patterns are regarded by them as arising by an integration of these units. Nevertheless, all the evidence that has accumulated from the study of the development of reflexes, and particularly from the studies by Bersot, definitely establishes that the reflex is in no real sense a unit.

Bersot [21] has made extensive statistical studies of the plantar reflex in development from fetal into postnatal life. He says that the external stimulation acts, not on one determined part of the organism, but on the general organic condition, which is variable; that the organism is interposed between the stimulation and the reaction; the different modes of reaction are not in immediate connection with each other, but in a connection determined by the general condition. This last statement by Bersot is corroborated by my observations on the development of the walking gait in *Amblystoma* where, as already noted, the reaction of one limb is not in immediate relation to the action of any one of the others, but the action of each limb is in direct relation to the trunk as a whole, and the action of the latter is the agency of integration of the limbs.

Elsewhere, Bersot [22] says that if one judges only by the motor reaction, after the plantar reflex has become differentiated, one will conclude that the stimulus which formerly acted on the entire organism now acts on the lower limb alone, but, he adds that is not so: the stimulus still continues to act on the entire organism. Again he [23] asserts that the plantar reflex of the infant shows one clearly the impossibility of considering reactions as varying independently one of another, and that each manifestation, the whole of which

21. Bersot (footnote 14, first and second references).

22. Bersot (footnote 15, third reference).

23. Bersot (footnote 14, fourth reference).

constitutes the plantar reflex, is then to be considered as an integral part of a whole which evolves in proportion as the organism develops. These conclusions of Bersot from his extensive and highly technical statistical studies of the development of the reflex are exactly in accord with the development of reflexes in *Amblystoma* as I have seen them, and they certainly refute the idea that the reflex as seen in physiology has that fixed individuality which characterizes a unit. They are also corroborated by the observations by Minkowski, who endorses them,[17] and asserts that in a certain sense and at least in latent form the whole organism participates in every reaction, so that what impresses one as a reflex constitutes only the especially manifest and visible form of it.

This interpretation of dominance of the organism as a whole over reflexes is further supported by the early development of postural reflexes in the human fetus as demonstrated by Minkowski, and by the observation by Bolaffio and Artom of shoulder movements repeatedly in a human fetus of 7 cm. in response to the prick of a pin in the cerebral cortex, and an increase in the strength of reflexes after ablation of the cerebral hemispheres in this same specimen. According to the latter observation, the cerebral cortex begins to participate in the behavior of man approximately with the beginnings of the oral reflex, the eyelid reflex and the grasping reflex of the toes, and while virtually a total reaction can still be stimulated by a touch on the arm or body (table 2).[24]

It appears, therefore, that from the beginning of the process of devolopment of reflex specificity of receptors and the individuation of reflex patterns, the cerebral cortex of man operates with the rest of the nervous system as a unit. Accordingly, if there is such a thing as a "unit reaction" in nervous function, it is the total pattern; and the development of specific nervous functions, such as reflexes of different grades, is an analytic process, not a synthetic one. This being true, any impairment of the integrity of the nervous mechanism as a whole, either experimentally or pathologically, must necessarily accen-

24. In the discussion of their observations Bolaffio and Artom seem to disclaim any evidence of cortical function during the first six months of fetal life. They say that the cortex (rolandic and other zones) and the pyramidal tract in the internal capsule and in the peduncles are unexcitable during this period. In this conclusion they are no doubt in accord with the prevailing opinion. Nevertheless, they say, also, that after decerebration they observe constantly an increase of the muscular reflexes, and they cite explicitly their cases number 2, of 7 cm.; number 5, of 23 cm.; number 9, of 33 cm.; and number 11, of 34 cm. So far as their evidence goes, it appears that decerebration has an effect on the behavior of fetuses of 7 cm. and older, and that, although the cerebral cortex may not be excitable to mechanical or electrical stimulation at this time, it must participate in the function of the nervous system as a whole.

tuate the analytic process, and bring subsidiary mechanisms out into a relation of relative dominance over a fragment of the system. The study of such subsidiary mechanisms has added incalculably to the knowledge of nervous functions; but their actual meaning, it seems to me, cannot be adequately revealed without exhaustive knowledge of their origin and development in the growing organism.

In conclusion, I am convinced, by a study of all available records of movement in human fetuses of the first 6 months, that behavior develops in man as it does in *Amblystoma* by the expansion of a total pattern that is integrated as a whole from the beginning and by individuation of partial patterns (reflexes) within the unitary whole. This principle of embryologic development of the function of the nervous system seems to me to have important bearing on fundamental problems of both normal and pathologic physiology and psychology.

THE ANATOMY OF THE DEPRESSOR NERVE IN MAN*

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In recent years considerable interest has been aroused concerning the rôle of the depressor nerve in angina pectoris and the possibility of relief through sectioning this nerve. As there is variance in the use of the term "depressor nerve," and as its anatomy is, in general, little understood, this study was made with a view to clarifying the matter.

REVIEW OF THE LITERATURE

In the literature, the name "depressor" has been given to several nerves of vagal origin, first of all to a nerve occurring in the rabbit, described first by Cyon and Ludwig.[1] This nerve has been shown repeatedly to be a depressor by physiologic experiments. Stimulation of the peripheral end of the cut nerve in the rabbit, as a rule, gives negative results, while stimulation of the central stump causes a reflex fall in blood pressure. The name has also been given to the cardiac branches of the vagus in man, as well as to other nerves coming from the vagus. It is doubtful if any of these nerves in man represent the depressor as it occurs in the rabbit.

The depressor nerve in the rabbit was first described as a fine trunk arising by two roots, one from the vagus and the other from the superior laryngeal nerve. It has a free course through the neck, running parallel to the vagus and the cervical sympathetic trunk, and is finally lost grossly in the cardiac plexus.

Some years later, Köster and Tschermak[2] dissected out the depressor nerve in fifty rabbits. Their observations on the origin of this nerve are given in the accompanying table.

These authors located the cells of origin in the upper pole of the jugular ganglion. Molhant[3] located the cells of origin in a restricted

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1. Cyon and Ludwig: Die Reflexe eines der sensiblen Nerven des Herzens auf die motorischen der Blutgefässe, Arbeit a. d. physiol. Anst. zu Leipzig, 1867, vol. 1.

2. Köster, G., and Tschermak, A.: Ueber Ursprung und Endigung des N. depressor and N. laryngeus superior beim Kaninchen, Arch. f. Anat. u. Physiol. (suppl.) 1902, p. 255.

3. Molhant, M.: Les ganglions périphériques du vague, Le névraxe **15**:525, 1913.

area of the ganglion nodosum. He described the depressor nerve in the rabbit as one consisting entirely of small myelinated fibers. (My count gave between 200 and 300 such fibers in his figure 19.)

Thus, there is good evidence that, in the rabbit, the depressor nerve, while varying considerably in its exact mode of origin, is practically constant in occurrence; it is composed of fibers of about the same diameter, and, as is clear from the location of its cells of origin and the results of electric stimulation, it is a purely sensory nerve.

In considering the situation in man, it should be remembered that the name "depressor" has been applied to several nerves. Both the superior and the inferior cardiac branches of the vagus have had this name attached to them. A communicating branch between the superior laryngeal nerve and the cardiac branches of the cervical sympathetic

The Origin of the Depressor Nerve in Rabbits as Observed by Köster and Tschermak

Depressor arising by two roots, one from the vagus, the other from the superior laryngeal nerve	29
Depressor from the vagus alone	14
Depressor from the nodosal ganglion	6
Depressor from the angle of the vagus and the superior laryngeal nerve	20
As above, but with two roots	1
Depressor lacking unilaterally	4
Depressor lacking bilaterally	1
Depressor from the superior laryngeal nerve alone	25

trunk has also been designated "depressor"; and a nerve in man having a gross anatomic resemblance to the depressor nerve in the rabbit has also been described and called the depressor nerve.

The last mentioned was first clearly described by Kreidmann,[4] who found a small nerve that arose by two roots, one from the vagus and the other from the superior laryngeal nerve, which, after a separate course of as much as 3 cm., was reunited with the vagus (fig. 1*A*). From the manner in which it came from the vagus and the superior laryngeal nerve, Kreidmann believed that it was the nerve that corresponds to the depressor nerve in the rabbit. He said that the nerve was of constant occurrence, although it was often necessary to dissect away the sheath of the vagus to find it, especially on the left side.

4. Kreidmann, A.: Anatomische Untersuchungen über den Nervus depressor beim Menschen und Hunde, Arch. f. Anat. u. Physiol. (anat. sect.) 1878, p. 403. (Kreidmann gives priority to M. Bernhardt, 1868, for first noticing this nerve in man.)

Finkelstein [5] found the depressor, as described by Kreidmann, in two of five cadavers. He described it as a nerve 1 mm. in diameter and 2.5 cm. in length running back into the vagus. Békésy [6] found the depressor nerve of Kreidmann in nine of fourteen cadavers. He again described it as a little nerve running in isolation for about 1 cm. and then recombining with the vagus.

Alpiger [7] dissected nineteen cadavers and found the nerve described by Kreidmann in only two cases. He also found a nerve issuing singly from the superior laryngeal nerve which flattened out against the vagus immediately. His opinion was that this was also the depressor nerve. He found one instance of a nerve arising like the depressor and taking a separate course to the heart. Alpiger described a communication between the superior laryngeal nerve and the superior cardiac nerve of the sympathetic. He assumed that the superior laryngeal nerve contained depressor fibers, and from that argued that the "ramus anastomoticus" to the superior cardiac nerve (sympathetic) is the usual form of the depressor nerve in man. When this arrangement was lacking, he found a strongly developed superior cardiac ramus from the vagus.

Viti [8] dissected 100 cadavers and found the depressor lacking on both sides seven times, lacking on the right side eighteen times and lacking on the left side twelve times. Viti said that he was unable to report a single type, but that the depressor nerve in man is represented by a branch of the superior laryngeal nerve, which goes either directly or indirectly to the heart.

Odermatt [9] cut the depressor nerve in a case of angina pectoris. In this case, the nerve that was cut arose from the vagus alone, below the origin of the superior laryngeal nerve. The sectioning of the nerve was followed by a rise in blood pressure.

Perman [10] said that in man he found a twig passing from the superior laryngeal nerve to the superior cardiac nerve (sympathetic) in a great number of cases. In a few cases, the superior laryngeal nerve gave a branch to the vagus; occasionally he was unable to find

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5. Finkelstein, A.: Der Nervus depressor beim Menschen, Kaninchen, Hunde, bei der Katze und dem Pferd, Arch. f. Anat. u. Physiol. (anat. sect.) 1880, p. 245.

6. Békésy, E.: Adatok a szividegek boncztanához, Orvosi hetil. **32**:20, 1888; cited in Zentralbl. f. Physiol. **2**:176, 1888.

7. Alpiger, M.: Anatomische Studien über das gegenseitige Verhalten der Vagus- und Sympatheticusäste im Gebiete des Kehl-kopfes, Arch. f. klin. Chir. **40**: 761, 1890.

8. Viti, A.: Sur le nerf dépresseur chez l'homme et chez les autres mammifères, Arch. ital. de biol. **5**:191, 1884.

9. Odermatt, W.: Die chirurgische Behandlung der Angina pectoris, Deutsche Ztschr. f. Chir. **182**:341, 1923.

10. Perman, E.: Anatomische Untersuchungen über die Herznerven bei den höheren Säugetieren und beim Menschen, Ztschr. f. d. ges. Anat. **71**:382, 1924.

a communication between the superior laryngeal nerve and the sympathetic or vagus.

According to Schumacher,[11] the depressor nerve is of constant occurrence in all mammals, and in man is represented by the cardiac branch of the superior laryngeal nerve and the upper cardiac branches of the vagus.

Tschermak[12] used the name "depressor" to designate all of the cardiac branches of the vagus, and the cardiac branches of the superior laryngeal nerve, as well. He had both an inferior and a superior depressor nerve coming from the vagus.

Hofer,[13] in a series of ten operations, found the "depressor" nerve on both sides in five cases, and in three more in which the nerve was sought on the left side alone, it was found and cut. In one case, the nerve could not be found on the left side, but a small one was found on the right side. In one case, the nerve could not be found on either side. In these cases, Hofer sought a nerve coming either from the vagus or from the superior laryngeal nerve. He stated that extensive dissection of the neck was necessary to find the nerve.

Fedoroff and Saposchoff,[14] as the result of a series of dissections, found six different ways in which the "depressor" nerve might arise. The most usual origin was from the external branch of the superior laryngeal nerve with the depressor running into the vagus after a short separate course. This type of depressor nerve they found in 50 per cent of their cases. These authors recommended that when a separate depressor nerve cannot be found, the vagus sheath be opened and the most medial fasciculus cut immediately beneath the nodose ganglion.

The preceding review of the literature brings out the following facts: (1) the name "depressor" has been given to various nerves that resemble one another only in the fact that they are of vagal origin; (2) little study has been made of the histology and physiology of these nerves in man, notwithstanding the fact that such a study is necessary if their function is to be established.

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11. Schumacher, S.: Zur Depressorfrage, Ztschr. f. d. ges. Anat. **75**:259, 1924.

12. Tschermak, A.: Ueber die afferente Innervation des Blutgefässystems, anatomisches, physiologisches, allgemeinpathologisches, Wien. med. Wchnschr. **74**: 899 and 958, 1924.

13. Hofer, G.: Zur Klinik und Technik der Depressordurchschneidung bei der Angina Pectoris, Wien. klin. Wchnschr. (suppl.) **37**:1, 1924; Wien. med. Wchnschr. **74**:1356, 1924.

14. Fedoroff, S., and Saposchkoff: Zur Technik der Operativen Behandlung der Angina Pectoris mit Durchschneidung des Nervus Depressor, Zentralbl. f. Chir. **52**:1937, 1925.

The nerves to which the name "depressor" has been given by the various authors may be grouped as follows: (1) the rami cardiaci superiores of the vagus; (2) a nerve having an origin like that of the depressor in the rabbit, but differing from the nerve in the rabbit in that after a short separate course it reunites with the vagus, and (3) a branch of the superior laryngeal nerve, which unites with the cardiac branches of the cervical sympathetic nerve. Which one of these, if any, is a depressor nerve in a truly physiologic sense as is the nerve in the rabbit (that is, a purely sensory nerve, stimulation of which causes a reflex fall in blood pressure) has not yet been determined.

There is, in fact, some evidence that at least one of these groups, namely, the superior cardiac branches of the vagus, is not purely sensory. Danielopolu,[15] from physiologic experiments on dogs, concluded that the inferior depressor nerve of Tschermak, as well as the branches cut by Hofer in his operations, contain pressor as well as depressor fibers.

ANATOMIC AND HISTOLOGIC OBSERVATIONS

In the anatomic laboratory at the University of Minnesota, I carefully examined sixteen cadavers for cardiac branches coming from the vagus below the origin of the superior laryngeal nerve. I found such branches eleven times on the left side and eight times (in thirteen cadavers examined) on the right side. The size of these branches varied from extremely fine filaments to branches from 2 to 3 mm. in diameter.

Pieces of these branches were sectioned and examined histologically. In all cases, they contained myelinated fibers of all sizes, ranging from large to exceedingly fine ones. The presence or absence of nonmyelinated fibers was not determined. The number of fibers considered large varied from 7 to 75, with smaller fibers in approximately the same proportion. Not only was this great variability found, but it was also impossible to judge the amount of nerve tissue present by the gross size of the branch. More than once a branch of considerable size was composed largely of fibrous connective tissue with only a few nerve fibers, while a fine branch, when sectioned, would be found to be composed of a fairly large number of nerve fibers closely packed together.

Since the superior cardiac branches of the vagus are so highly variable in their point of origin from the vagus (coming off anywhere above the clavicle, as examination of a series of anatomy textbooks will show) and vary so greatly in the number and the size of the nerve

15. Danielopolu, D.: Sur l'existence d'un nerf presseur-dépresseur chez le chien. Considérations sur le traitement chirurgical de l'angine de poitrine, Compt. rend. Soc. de biol. **95**:112, 1925.

fibers that they contain, and since at least one investigator (Danielopolu) produced evidence that they contain pressor as well as depressor fibers, it seems improbable that they correspond to the depressor in the rabbit. Hence the term "depressor" should not be applied to them until more reliable evidence is produced. The term given them in the

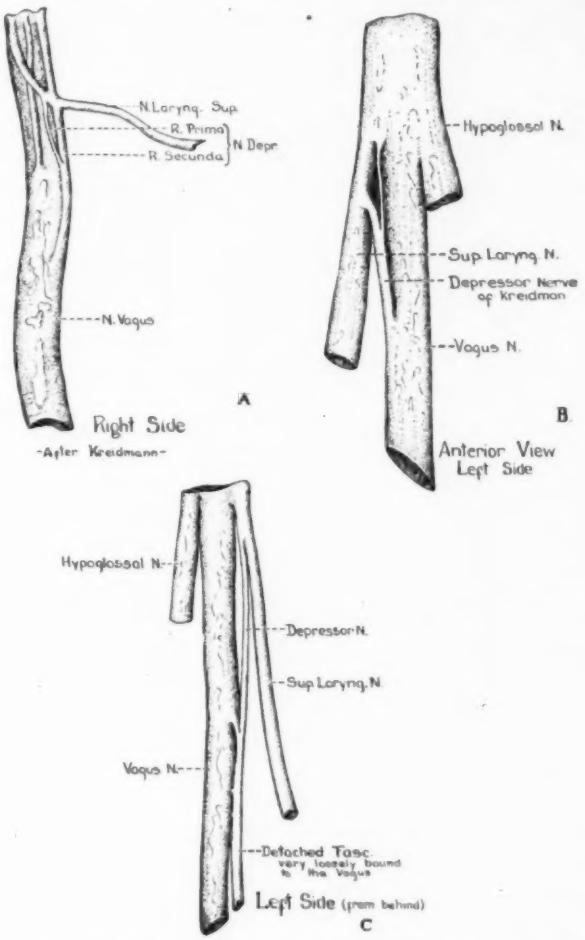

Fig. 1.—*A* shows Kreidmann's "depressor nerve." It is shown arising by two roots, one from the vagus and the other from the superior laryngeal nerve; *B*, a nerve or fasciculus similar to that of Kreidmann, found in gross examination in two of six cadavers that were dissected on the left side, and *C*, a fine bundle of fibers resembling Kreidmann's "depressor nerve" found in two of six cadavers that were dissected on the left side.

"Basle Nomina Anatomica", rami cardiaci superiores, would seem to be preferable at present.

In a later series of dissections, the nerve of Kreidmann was sought. In six dissections on the left side, a nerve or fasciculus similar to that described by Kreidmann was found well developed in two cases (fig. 1*B*); fine bundles of the same nature were present in two more cases (fig. 1*C*) and in the other two none was visible. These results were obtained without dissecting the sheaths surrounding the nerves. In a different set of cadavers six dissections were made on the right

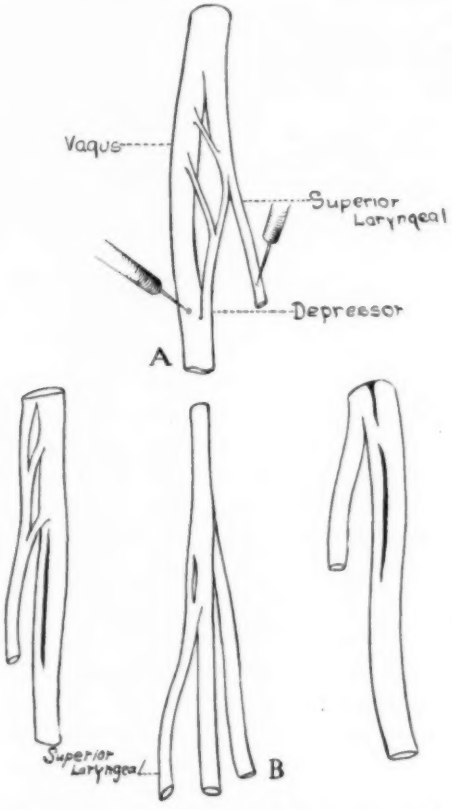

Fig. 2.—*A*, the "depressor nerve" of Kreidmann found after removal of the nerve sheaths; *B*, other ways in which the superior laryngeal nerve leaves the vagus (nerve sheaths removed).

side. Only one short but definite nerve of Kreidmann was found without dissection of the vagus sheath. An additional one was found by extensive removal of the epineurium from all the nerves concerned (fig. 2). Longitudinal sections of the vagus, including the first part of the superior laryngeal nerve, were made of two of the specimens from the right side. One was the specimen in which the nerve of Kreidmann was visible without the removal of nerve sheaths. The other was a specimen in which such a nerve was not visible.

Examination of the sections showed, in the first case, a bundle leaving the superior laryngeal nerve, and this, joining with a smaller bundle of fibers from the vagus itself, ran back into the vagus again after a short separate course (fig. 3). This was the specimen in which the depressor of Kreidmann was visible. The other specimen showed a similar phenomenon, which had been entirely concealed by the connective sheaths of the nerves in the gross examination. Serial cross-

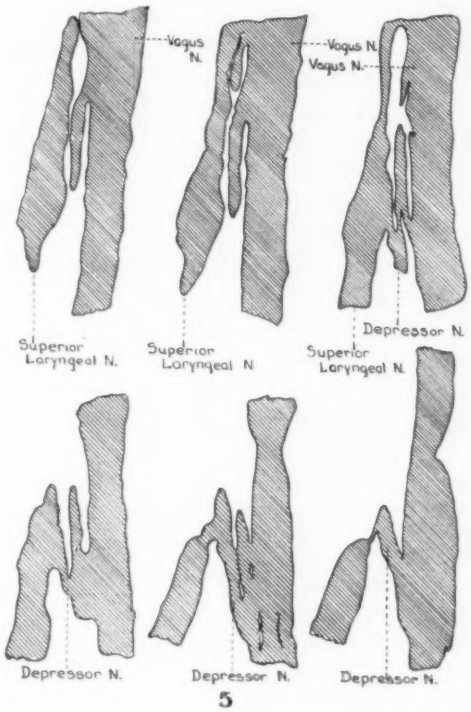

Fig. 3.—Projectoscope drawing to show the "depressor nerve" of Kreidmann. The drawing is made from the longitudinal sections of the right vagus and the superior laryngeal nerve.

sections were made of two specimens from the left side. Here, also, one with a visible nerve of Kreidmann and one without were taken.

An examination of the cross-sections from the specimen showing a nerve of Kreidmann grossly revealed a rather large fasciculus from the superior laryngeal nerve, that ran into the vagus at nearly the same point at which two more or less detached fasciculi of the vagus united with the main bundle. The vagus then continued downward for several millimeters as practically one fasciculus, except for a small bundle composed of fibers of all diameters lying at one side of the main trunk.

This small bundle ran into the vagus at a still lower level; a part of it came directly from the vagus above, where the communication from the superior laryngeal nerve joined the main trunk. Other portions of this same small bundle were rather indefinitely traced to both the accompanying hypoglossal nerve and the superior laryngeal nerve. While this small fasciculus seemed to contain a greater proportion of large fibers than was usually found in the vagus proper, it was certainly not a bundle of fibers of uniform size. The large connecting fasciculus between the superior laryngeal nerve and the vagus presented the same picture as the two nerves themselves; that is, fibers of all sizes without any tendency for the various sizes to be collected into different groups. The other specimen, when examined in cross-sections, showed four small fasciculi from the superior laryngeal nerve, which, after communicating with each other, joined the main trunk of the vagus. At a level slightly lower than that of the origin of the superior laryngeal communications to the vagus, the vagus itself gave off a small bundle composed most exclusively of large medullated fibers. This bundle ran with the superior laryngeal nerve as far as the sections went without being intimately joined to the nerve. This last mentioned bundle of fibers might be considered of some significance on the ground that it consisted almost wholly of large fibers, if it were not for the additional fact that no trace of this bundle of fibers was seen in the three other specimens examined microscopically.

In one set of cross-sections, the vagus immediately below the ganglion nodosum consisted of one large fasciculus and one small one; in the other it was composed of several medium-sized bundles and several small ones. Such an observation is to be expected from the work of Barratt,[16] who long ago pointed out that the fasciculi in the vagus do not have a definite pattern but change in their form and arrangement every few millimeters in the region in which the superior laryngeal nerve is given off. When such is the anatomy of this region, it does not seem reasonable to cut the most medial fasciculus of the vagus immediately beneath the nodose ganglion as Fedoroff and Saposchkoff recommended in case a separate depressor cannot be found.

CONCLUSIONS

The question of a separate depressor nerve in man, that is, a purely sensory nerve with an ultimate distribution such as is found in the rabbit, may be summed up as follows:

1. A nerve resembling the depressor in the rabbit in all respects grossly is found rarely (Alpiger, 1 case; Fedoroff and Saposchkoff, 1 figure).

16. Barratt, W.: On the Anatomical Structure of the Vagus Nerve, J. Anat. & Physiol. **32**:422, 1898.

2. Evidence contrary to the claim of a purely depressor nature for the superior cardiac rami of the vagus has been presented. These branches are extremely variable as to size, number and position.

3. The ramus between the superior laryngeal nerve and the cardiac branches of the sympathetic nerve is of inconstant occurrence, and its claim to being the depressor nerve is based on the assumption that the superior laryngeal nerve regularly carries the depressor fibers. Against this assumption is the fact that the depressor in the rabbit may often come from the vagus alone.

4. The depressor nerve of Kreidmann occurs on one or both sides in a considerable number of cases, but varies greatly in size and in exact relations. The histologic evidence, though limited, seems to show that it is only a more detached portion of the intraneural plexus of the vagus.

5. None of the nerves described has been shown to be a depressor nerve physiologically.

The statements thus made do not mean that I wish to convey the impression that it is impossible to locate and cut cardiac branches of vagus origin, or that such a procedure might not be beneficial in cases of angina pectoris; I merely wish to point out that in man there is not a well defined nerve at any point from the exit of the vagus from the skull to the level of the clavicle that has been proved to be a depressor nerve. Severing these nerves probably does more than remove a certain amount of sensibility, and, because of the great variability of the vagus branches to the heart, variable results are to be expected from such an operative procedure.

CEREBRAL CALCIFICATION EPILEPSY

ENDARTERITIS CALCIFICANS CEREBRI *

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The coincidence of epilepsy and cerebral calcification is not a new observation. However, in this communication we are dealing with epilepsy of a peculiar type that appeared in one family, including the father and all four children. This, together with the fact that there was in each case also a peculiar type of cerebral calcification, justifies the assumption that we are dealing with a hitherto undescribed disease entity. There is strong evidence that the pathologic process in each case is primarily in the arterioles and capillaries of the cerebrum and presumptive evidence that the epileptic attacks have as their cause a vasomotor mechanism.

REPORT OF CASES

It seems best to present each member of the family in turn, although it has not been possible to study them all as completely as the patient in case 3.

CASE 1.—*History.*—Ev., a girl, aged 15, had been delivered at term with instruments and was apparently normal as a child. She seemed mentally normal up to the age of 2 years. She began to talk at 1 year and walked at 26 months. After this time, according to the mother, her mental development "slowed down." At 8 years of age, she could play about, keep herself clean, sew on buttons, etc., but seemed unable to learn anything new. Catamenia began at 12 years. She now is a "mirror writer." She is left-handed, can write her name, but little else, and draws pictures at times to explain what she cannot say. For the past eight years, she has been confined in the New Jersey State Sanatorium for Epileptics.

She had periodic enlargements of the glands in both sides of the neck. She had measles, chickenpox, influenza and tonsillitis. Tonsillitis or any infection increased the number of convulsions. She complained of headaches.

Convulsive Attacks.—When the child was 6 weeks of age, the mother noted that she was "restless and on the move," and there were observed periodic twitchings of the right arm, contractions of the pupils and a pulling of the mouth to the left side. Between 6 and 8 months of age, she moved her right arm but little, and she would not lift it. This disability disappeared, but she became left-handed.

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* From the Departments of Medicine and Surgery of Columbia University, and the Presbyterian Hospital.

* H. R. G. is responsible for the original study of this remarkable family. W. P. is responsible for the surgery, pathology and comment.

When she was 14 months old, the epileptic convulsions seemed to reach their height. There were several attacks a day, characterized by thrusting of the right leg and occasionally by loss of sphincter control. One bilateral convulsion lasted for ten hours. From the age of 2 to the time of the present study, the patient had only five or six severe convulsions, although the minor attacks continued. During the seizures, the face did not become blue, and sweating was not pronounced, but the attacks were always accompanied by contraction of the pupils. At the time of the present study they consisted only in a twitching of the left side of the face, associated with pupillary contraction.

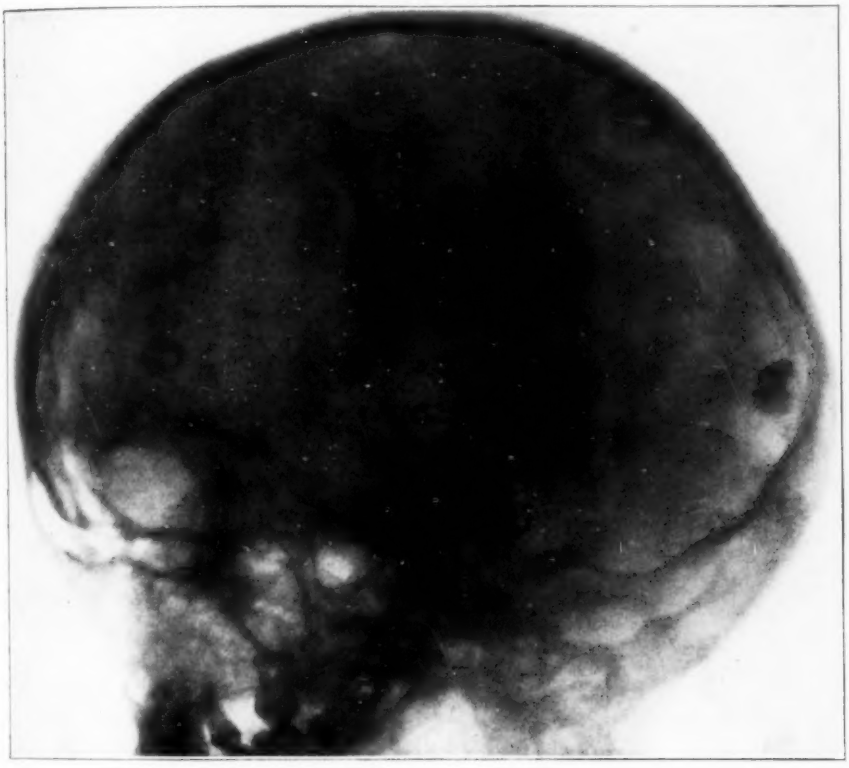

Fig. 1 (case 1).—Discrete calcified areas in the brain.

We were unable to examine this patient personally. Her condition seemed at the time of this study to be stationary, and she was enjoying physical well-being.

Roentgenograms.—Through the kindness of Dr. David Weeks, of Skillman, New Jersey, roentgenograms were taken of the patient and sent to us. They showed discrete calcified foci within the brain, many of which gave the impression of being hollow (fig. 1).

CASE 2.—*History.*—H., a boy, born normally three years after the patient in case 1, seemed to develop unusually rapidly up to the age of 18 months, after which the advance ceased. He spoke connected words at 10 months. At 14 months, he could sing correctly part of a song that he had heard a neighbor sing. He crept

at 10 months and walked at 3½ years. He was right-handed. He was subject to attacks of diarrhea. According to the mother, he occasionally had swelling of all the lymph glands, but this did not have any relation to the convulsions.

Convulsive Attacks.—Beginning at the age of 5 months, he suffered from generalized convulsions, and from the age of 8 or 9 months there were smaller attacks as well. The convulsions became steadily worse, reaching their height at the time of his death at 4 years of age in what seems to have been status epilepticus. These attacks were accompanied by loss of sphincter control. During the last two months of life, the patient sometimes had syncope without a convulsion. The pupils were invariably dilated during the fits, and the face became purplish. No particular sweating was observed. Toward the end of life, the pupils were continously large.

Roentgenograms were not taken, so far as can be discovered, but an autopsy was obtained in the hospital at Elizabeth, N. J., and a report of "Multiple Calcified Tumors of the Brain" was made. Unfortunately, the pathologist who made this report subsequently died, and the specimen was lost, so that this diagnosis could not be verified.

Case 3.—*History.*—A, a boy, aged 10, had been born normally at term. His mental development up to the age of 2 years seemed at least as good as the average. From that time on, however, he was definitely retarded, although he had talked at 1 year and had begun to walk at 14 months. He was right-handed.

At 2 years, he had, the mother believed, a swelling of the glands of the neck during attacks. Later, it seemed to her that the glands became swollen before a severe attack. At the age of 4, during a period of months, he slept little, never longer than fifteen minutes at a time.

Convulsive Attacks.—Four days after the child's birth, the mother noticed that at every feeding the left side of the child's face underwent a certain degree of spasm. The facial muscles seemed to be "drawn downward on this side, and both pupils were contracted" to a degree that she described as a "pinpoint." These contractions, which were present at every feeding, were transient, lasting not more than one or two seconds. This type of attack continued until the child was 2 months of age.

After the infant had been shifted from breast to cow's milk, diarrhea developed and lasted for three days; the child then had a generalized convulsion. This type of convulsion continued to recur at intervals until the time of the present study. It was described by the mother as follows: Without cry or any discoverable aura, the patient's right upper extremity began to twitch, usually starting with the hand. After this had obtained for a few seconds, the arms, legs, head and body went into a generalized tonic-clonic spasm that lasted for from two to four minutes. Biting of the tongue and frothing at the mouth were not present, and when the convulsion stopped the patient went to sleep. The faciopupillary spasms and the generalized convulsions continued, and small attacks involving the right arm and the left side of the face were of daily occurrence up to the age of 9. The daily number of the minor attacks averaged fifty or more, while there were often from one to four of the major attacks a day.

At this time the child, aged 8, was sent to school at the New Jersey State Sanatorium. His condition became so much worse that he had to be brought home at the end of a year. In his ninth year, the child was in status epilepticus for twenty-four hours, but improved and soon after, in January, 1925, the generalized convulsions ceased until the day of admission to the hospital, in October, 1925.

He continued to have the small attacks and there appeared a third type of seizure, that is, the head was suddenly turned to the left (rarely to the right), and, coupled with this, the right hand twitched while the face became flushed. This condition lasted several seconds, and then, as the spasm relaxed, the child became highly excited and burst into song or peals of laughter. At times, during this third type of seizure, the left eye was rotated inward.

For five years just previous to the present study, the mother had also noted that, when the patient walked, the right leg was thrown forward, the tip of the shoe scraping the ground as the foot traveled forward, so that this portion of the shoe had to be repaired at frequent intervals. During the forward movement, a striking inversion of the foot occurred.

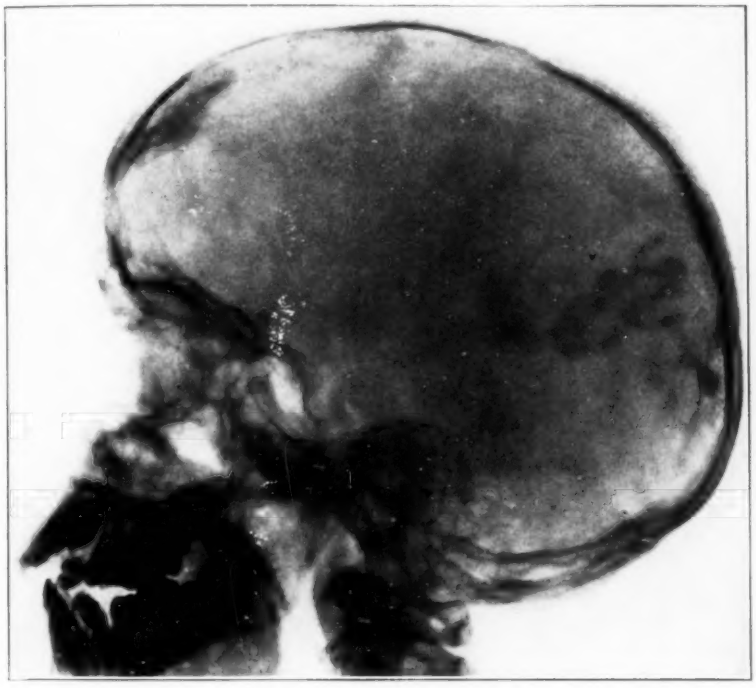

Fig. 2 (case 3).—Large calcified area in right occipital lobe.

At no time during the course of the described spasmodic conditions did chloral, phenobarbital or bromides offer any striking relief, although these drugs were administered in full doses over periods ranging from weeks to months.

Examination.—The boy was fairly well nourished and well developed. His weight was 26 Kg., his height, 101.2 cm. He did not appear ill, and was affable and cheerful, but of low grade intelligence. The head was 53 cm. in circumference, and was flattened laterally. Tenderness was not present over the vault of the skull, mastoids or sinuses. The heart, lungs, abdomen, thyroid, genitals and extremities showed nothing abnormal. The blood pressure was: systolic, 100, and diastolic, 62.

The neurologic examination did not point clearly to a localized lesion. There was paresis of the left external rectus, however, and the left pupil was larger than

the right. Both pupils reacted promptly to light and on accommodation. At times there was spontaneous nystagmus to the left; also, occasionally, to the right. The corneal reflexes were both present, but definitely less active than in the normal eye. The optic disks showed some primary atrophy. The deep reflexes were symmetrically active, and there was bilaterally a transient ankle clonus. The abdominal reflexes were active and equal and the plantar responses normal. The sensory examination, as far as the patient's mentality permitted it to be carried out, yielded nothing abnormal. It was not possible to test coordination or visual fields, or to make other more intricate tests because of the boy's lack of cooperation. Mentally, he was definitely deficient, not understanding the simplest commands; when asked to put out his tongue, he opened his mouth or did nothing.

Roentgenograms.—A large calcified egg-shaped area was observed in the right occipitoparietal region (fig. 2). When stereoscopically studied, this mass seemed partly hollow. Posterior to this larger area, some scattered calcification was seen in the region occupied by the occipital lobe. Farther forward there was a small nodule, resembling in size and position a calcified pineal gland, except that it was displaced across to the left side 1 or 2 cm.. Roentgenograms of the thorax and the upper part of the abdomen did not reveal any evidence of calcification in these regions.

Special Examinations.—The red count was 5,000,000; the hemoglobin content, 80 per cent, and the white cell count, 10,000. The differential white count was normal, and the leukocytes were of normal appearance. The basal metabolic rate was minus three (Dubois). The Wassermann reaction of the blood was negative. The blood calcium was 9.8 mg. per hundred cubic centimeters.

Treatment.—The patient was kept fasting for eight days and then was placed on a ketogenic diet until the time of the operation. This regimen seemed to be accompanied by a slight diminution in the number and the intensity of the convulsive attacks, although a striking change for the better was not noted. The operations on this patient are described below.

CASE 4.—*History.*—El., a girl, aged 8, was unusually intelligent during her first year, talking at 12 months and walking at 13 months. At the age of 2 years, her progress was much retarded, so that she never exceeded the mental age of 3 years. She was right-handed. She never had diarrhea, swelling of the glands or a serious illness.

Convulsive Attacks.—At about 5 or 6 months of age, during the eruption of her first tooth, the child began to have generalized convulsions. At 8 or 9 months, she started having minor convulsions. The attacks usually began in the left side, the left arm moving throughout more than the other extremities. The convulsions reached their maximum when she was 2 years of age, from four to five generalized convulsions developing daily, accompanied by cyanosis and loss of sphincter control. The attacks were always accompanied by pupillary dilatation, as in case 2. Sweating of the face was not noticed.

Recently, the attacks were no longer generalized. They would begin with lifting of the left shoulder. She would then run backward and stoop over to the right, but not fall.

Physical Examination.—A general examination revealed nothing abnormal, and, as in case 3, this patient presented few abnormal signs on neurologic examination. She had a left external strabismus that came and went spontaneously, being present at times for several days. The upper abdominal reflexes were gone, but the lower ones were present. Otherwise, she was physically normal.

Fig. 3 (case 4).—Calcified areas scattered through the more superficial portions of the cerebrum.

Mentally, she was either a low grade imbecile or in the upper levels of the idiot group. She was graded as below 3 years of mental age after tests at the Vanderbilt Clinic.

Roentgenograms.—The roentgenograms showed the most extreme degree of cerebral calcification. Collections of calcium were scattered through the brain (fig. 3). The deposition had spared, in general, the basal portions of the cerebrum and had left the cerebellum entirely untouched (fig. 4). As in case 3, many of these discrete foci of calcification appeared somewhat circular or tubular. Stereo-

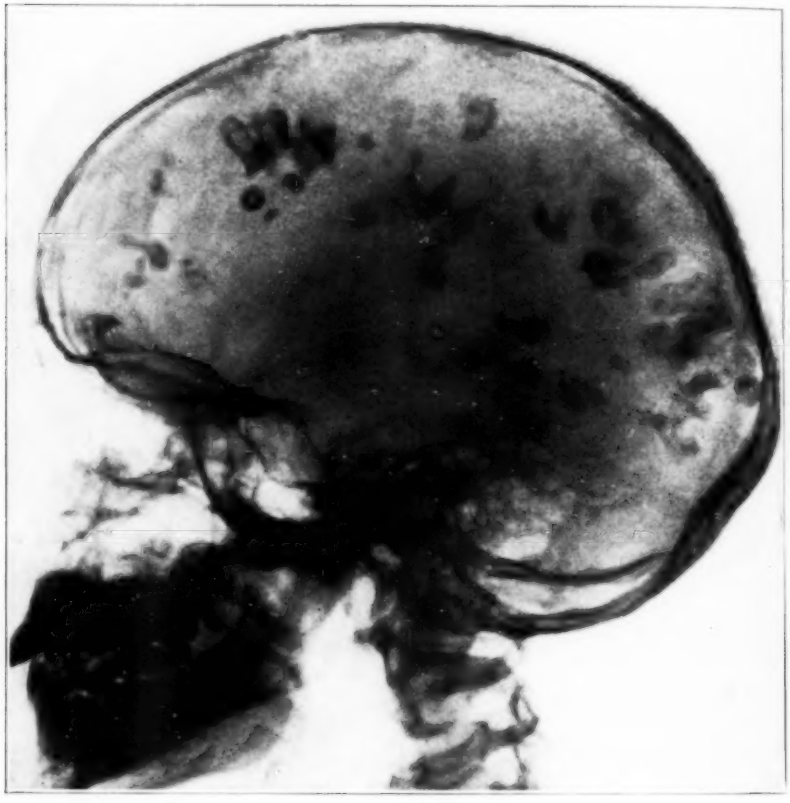

Fig. 4 (case 4).—The furrows that follow the fissures should be noted.

scopic study of the plates showed that the calcifications were for the most part near the surface of the brain, that is, removed as far as possible from the circle of Willis.

Case 5.—*History.*—The father, aged 48, was in good general health except that since early childhood he had been a sufferer from periodic attacks of migraine. He had been able to support his family and care for a green-house. One morning, eighteen months before the present study was made, he had three consecutive major epileptic attacks in which he lost consciousness, frothed at the mouth, bit his tongue and had a generalized clonic convulsion. He then fell asleep for an hour and

awoke without any recollection of the untoward happening. After that time, he did not show any further evidence of epilepsy and, what is more, the migraine seemed to have disappeared.

This patient was not aware that he had had these convulsions, his wife having kept the matter secret. We therefore did not have an opportunity to examine him physically, but roentgenography demonstrated that he had a condition of the brain similar to that of each of his children. The roentgenograms showed calcified areas in the parietal and occipital lobes (fig. 5). At least one of these collections could be seen, stereoscopically, to be umbilicated or hollow. There were also a number of scattered intracerebral granules.

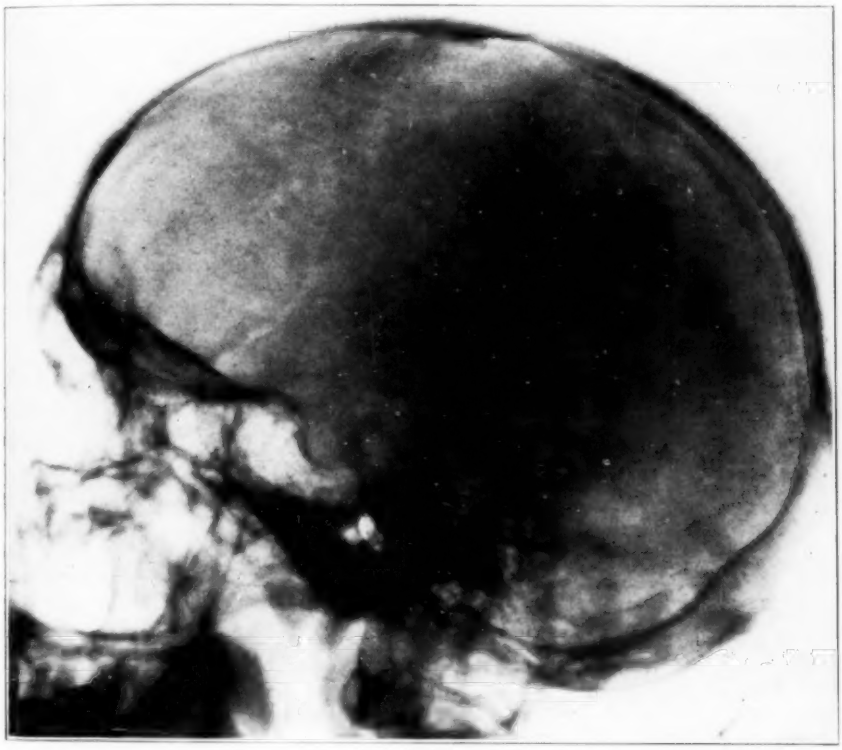

Fig. 5 (case 5).—Calcified areas in the cerebrum.

FAMILY HISTORY

As has been stated, all the immediate family, except the mother, were sufferers from the condition under consideration. The mother, aged 40, had always been strong and well. She had a high degree of intelligence, and she was a trained nurse. It was owing to her discriminating observation that we had so clear an account of the convulsions in each of the other members of the family. She stated,

however, that during her fourth and last pregnancy she had had what was called "puerperal insanity." Both the father and the mother were of Danish descent. They were born in neighboring villages near the coast of Denmark, where inbreeding is said to be frequent. In the mother's home village there was a high percentage of feeblemindedness. One of the mother's sisters had tuberculosis of the joints. Otherwise, there was no history of physical or mental disease in the antecedent or collateral members of the family, and no record of epilepsy, migraine, asthma or urticaria.

OPERATIONS IN CASE 3

In consideration of the treatment of our original patient, in case 3, it was at once evident that we were dealing with a familial condition and that the disease process could not be completely removed by operation.

In all the children, mental capacity was as good or better than normal during the first months of life. None of them showed obvious physical abnormality. All had suffered from a pathologic process that early caused epileptic convulsions and progressive mental deterioration. The site of this process was as obvious in the roentgenograms as it was in the history. The nature of the pathologic lesion, however, was not so apparent. The boy's mentality (case 3) had been much better than that of the other children up to a certain point; then he had become rapidly worse. Roentgenography (fig. 2) showed that in his case the process, whatever its nature, was localized. The specimens from the necropsy in case 2 had been lost, so that it seemed necessary to give credence to the pathologic diagnosis of calcified tumors in that case at least. It seemed that, if we really had to do with congenital tumors, they must be of slow growth. The mother was extremely anxious for an operation. An exploration was therefore finally undertaken in November, 1925, but with a dubious prognosis.

First Operation.—A bone flap was turned down over the right occipital lobe. The dura was rather tense and did not pulsate. It felt resistant, as though the underlying brain was hard. When the dura was reflected, the cortex was seen to have preserved its usual contour, but there was an area, about 7 cm. by 4 cm. in extent, in which the surface vascularity was obviously decreased, and toward this relatively avascular area the surrounding pial vessels converged, growing smaller as they approached it (fig. 6). On palpation, this area was found to be much harder than normal. The induration was uneven, apparently owing to the fact that the gyri were not equally hardened throughout. Posteriorly there were one or two slightly indurated areas, which apparently corresponded to the small calcified nodules seen by roentgenography to be in the occipital pole, posterior to the main mass. Anteriorly, the brain appeared to be normal, and one could feel a rather sharp transition from the anemic hard surface to the normal soft brain.

The whole occipital lobe was therefore resected. Hemorrhage was controlled with silver clips on the larger vessels and muscle on the smaller. It was found

that the induration extended well up to the median longitudinal fissure (fig. 6) but was not present at the base of the lobe. The dura was closed and the cavity filled with physiologic sodium chloride solution. Figures 7 and 8 are from roentgenograms taken after the operation to show the absence of the calcifications. The area removed is outlined by the hemostatic silver clips.

One calcified area persisted, as shown in these plates. This had been considered before operation as possibly a calcified pineal gland displaced to the opposite side

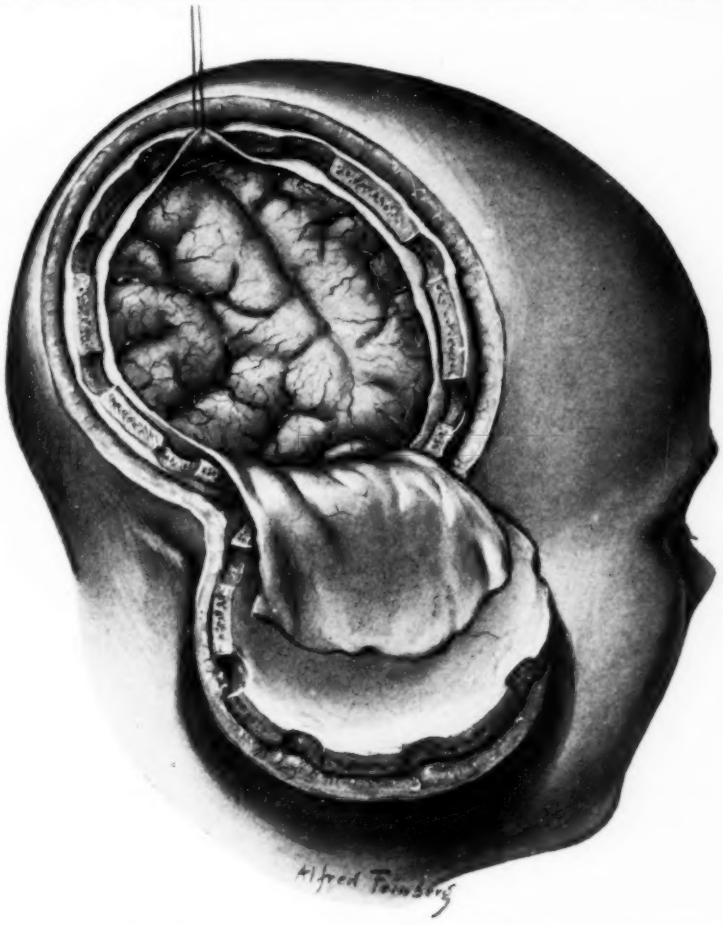

Fig. 6 (case 3).—Craniotomy, showing right occipital lobe. The anemic area corresponds to the calcified zone shown in figure 2. More bone was "rongeured" away in the lower anterior angle to make possible a complete removal.

by the mass in the parieto-occipital lobe. However, in the light of subsequent histologic observations, this had to be considered as a small area within the left parietal lobe.

Course.—The postoperative course was gratifying. During the first ten days, the patient was restless and often shouted aloud for little, if any, reason. After that, he became quiet and began to ask innumerable questions about all that went

on about him. On the fifth day, examination showed the following: the left ankle jerk was a little more active than the right, and the left abdominal reflex was slightly decreased; the plantar and cremasteric reflexes were normal. On the twenty-first day, he went home walking and with a new interest in the world about him.

He had then, of course, a left homonymous hemianopia. He had still a variable left abducens palsy and a slight weakness of the left facial nerve in voluntary but not in emotional movements. The deep and superficial reflexes were all normal and equal on the two sides.

Fig. 7 (case 3).—After the operation, one calcified shadow remained, which proved to be in the other hemisphere. Silver clips were placed on vessels.

PATHOLOGY

Macroscopic Appearance.—The solid tumor-like object that had seemed to be embedded in the excised brain was, in fact, not neoplastic but was a circumscribed alteration of the cerebral tissue. The indurated area was 8 cm. long, 3 cm. in one transverse diameter and 4 cm. in the other. It lay 5 cm. anterior to the extremity of the occipital pole and extended up to the midline, including several of the gyri that border the median longitudinal fissure. This median end of the mass was not so stony hard as other portions, but was firm and rubbery. In color this

portion resembled pink coral with a shade of tan. The major portion of the mass was hard, and the number of the overlying pial vessels was much reduced. Posterior to the mass, the surface of the occipital lobe appeared normal (fig. 9).

When the indurated area was cut through, the knife encountered a grating resistance like soft sandstone, but the brain markings were not obliterated. In cross section, a calcified zone was seen, shaped like a horseshoe, embracing a cortical fissure (fig. 10). This sandy zone was placed at about the junction of the white and the gray matter, and between the calcification and the underlying white matter was a translucent tissue, which was hard but not grossly calcified.

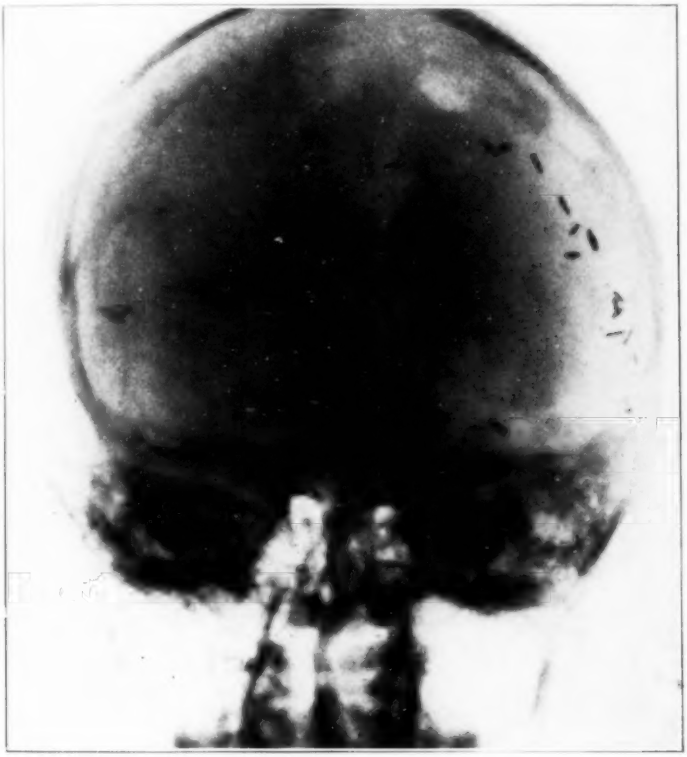

Fig. 8 (case 3).—The silver clips on the blood vessels outline the excised lobe.

Microscopic Appearance.—Histologic examination revealed nothing abnormal in the pia-arachnoid membrane. The blood vessels in portions of the brain remote from the hard area did not appear remarkable. In the occipital pole there was a zone of multiple perivascular hemorrhages in the deeper layers of the gray matter, and occasionally extending down into the white matter. These hemorrhages doubtless took place during the operative removal, but it may be significant that their location (fig. 11) in the uncalcified part of the brain roughly corresponded to that of the maximal calcium infiltration in the brain farther forward. Scattered through the more or less unaltered brain, particularly in the white matter, were typical corpora amylacea situated close to the small vessels (fig. 12).

In the indurated area, calcification began in the deeper layers of the gray matter and extended into the white matter. In the center of the calcified zone were loosely arranged fibrous neuroglia cells and many scattered calcified granules, which varied in size from less than the diameter of a nerve cell nucleus to the size of a Betz cell. The small capillaries stood out and seemed to be numerous, but were frequently converted into calcified tubes.

There were collections of calcium about larger vessels, both arteries and veins, in the form either of solid casts or of discrete granules. The vascular media was often thickened, and the deposits of calcium most frequently occurred in this layer. The intima also was sometimes thickened. A complete ring of calcium

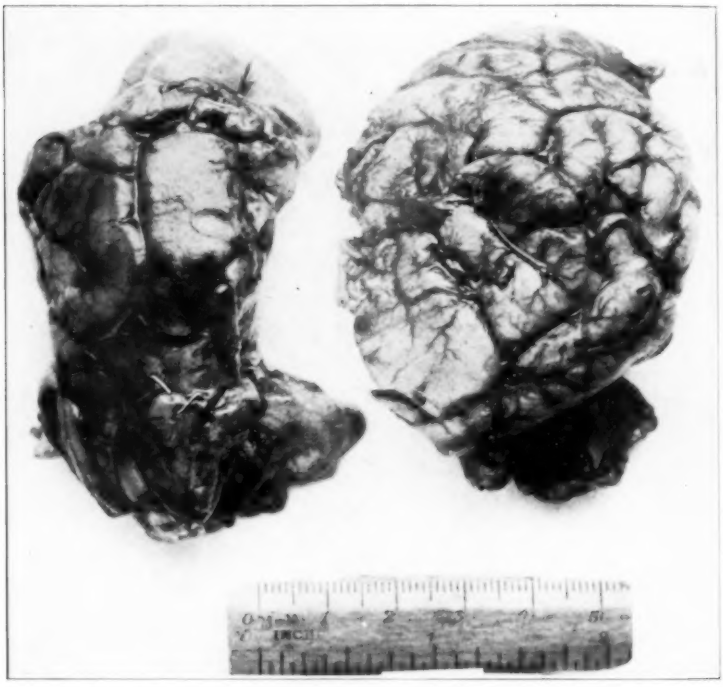

Fig. 9 (case 3).—The right occipital lobe in two segments.

might be present in the adventitia associated with much thickening of the media, as in figure 13. Thus, the vessel lumen was often found decreased down to the vanishing point (fig. 14).

In the avascular cortex, which has already been described as firm and of coral color, nerve cells were present, though much reduced in number. In some areas, the remaining cells appeared normal and satellitosis was not observed; while in other areas considerable pyknosis and elongation of the nerve cells were seen.

Changes of great interest were seen in the astrocytes. The superficial or zonal glia formed a layer considerably thicker than usual. Scattered localized areas of gliosis were seen in which the cells were hypertrophied and the expansions were fibrous and curly (fig. 15). Such groups were sometimes obviously arranged

around individual vessels, as though a decrease in the blood supplied by the vessel was responsible for the change (figs. 15 and 16).

In many areas of the brain, the protoplasmic astrocytes of the gray matter were perfectly normal. Nearer the abnormal zones, and especially in the "coral" area of the cortex and about the calcification, the proliferation of astrocytes was great, and many giant cells were seen, the cytoplasm of which was greatly enlarged, though the gnarled expansions of the cells tended to decrease in number. The

Fig. 10 (case 3).—A gross cross section of the indurated portion of the brain. The calcification may be seen in a zone surrounding a fissure, about which it forms a furrow.

Fig. 11 (case 3).—Tissue from the occipital pole at some distance from the calcified area.

nuclei of these cells were single or multiple and often of bizarre shape, and frequently were found at the cell margin (fig. 17). Some of these giant cells had progressed to evident degeneration and about them microglia cells were gathered in the process of dendrophagocytosis.[1]

1. Penfield, W.: Microglia and the Process of Phagocytosis in Gliomas, Am. J. Path. **1**:77, 1925.

The reaction of the neuroglia astrocytes was typical of that which follows progressive decrease in the supply of blood to an area of the brain, and the patchy arrangement of the gliosis, frequently centering about a small occluded blood vessel (as in fig. 16), suggested that the change in the vessel was the primary lesion.

The pathologic process in the brain was evidently an exceedingly chronic one. It was not primarily parenchymatous. The nerve cells were, for the most part, normal, except in the immediate vicinity of the calcified area. Even the astrocytes of the brain at a distance from the lesion did not show any abnormality, or diffuse gliosis such as

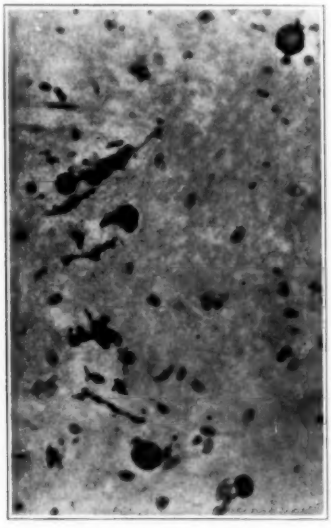

Fig. 12 (case 3).—Corpora amylacea in the brain at a distance from the chief calcified area.

occurs in toxic and infective processes. The primary change was a localized vascular one, but it was not in the larger vessels. Obliteration had taken place in the small vessels. There was local degeneration of cerebral tissue and, one may assume, the calcification was secondary to this local destruction, as it is well known that chronic local degeneration in any tissue may become the site of calcification.

As has been pointed out, the maximal degeneration and calcification were present at about the junction of the gray and the white matter. The cortical gray matter is said to be supplied, for the most part, by short arterial twigs that pass inward from the larger vessels in the pia mater. These arteries of supply should therefore be smallest at the junction of the gray and the white substance. It seems likely, there-

Fig. 13.—Perivascular calcification in the zone of greatest calcium deposit (hematoxylin and van Gieson stain).

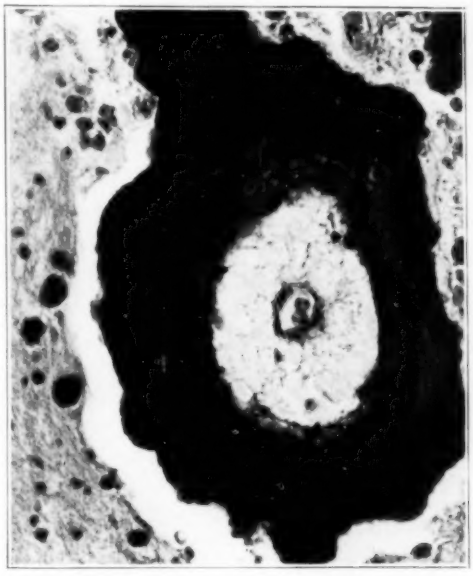

Fig. 14.—Thickening and calcification of a small vessel in the same area as shown in figure 13.

fore, that the process had taken place in the terminations of these arteries and in the capillaries and small vessels associated with them. The process might be called an obliterating endarteritis of the cerebral hemispheres.

Postoperative Course.—Following the operation in case 3, the patient's first month at home was the "best of his life," according to his mother. He made rapid advances mentally and did not have any

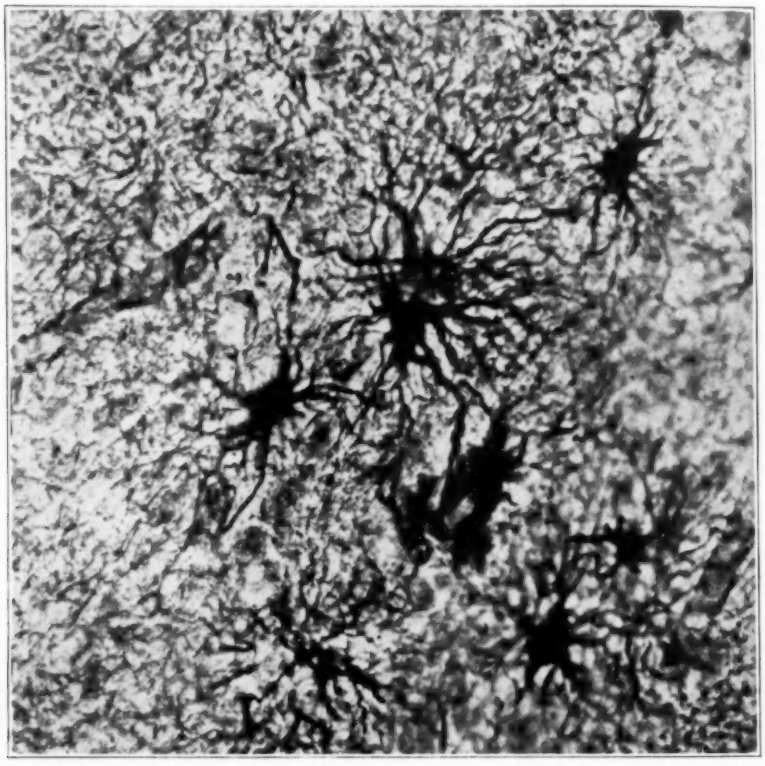

Fig. 15.—A patch of enlarged glia about a closed capillary (Cajal's gold chloride sublimate stain).

more epileptic seizures. He was examined by Dr. Robert B. McGraw, who rated his intelligence quotient at 55 and his mental age at 5½ years.

However, one year after the resection of the occipital lobe, he was brought back to New York because the attacks had begun to return and he was also a little less active mentally. The convulsive seizures were still associated with contraction of both pupils. At times, the right arm was the most involved, and, at times, the left arm and leg, with a

turning of the head to the right. There were two generalized convulsions associated with an attack of influenza.

Because of the evidence, in this case, that the pathologic process was in and about the small cerebral vessels, and because of the invariable association of the "fits" with pupillary change, it was decided, in September, 1926, to interrupt the vasomotor nerves going to one side of the brain, in the hope that this would hinder the evident progress of the disease.

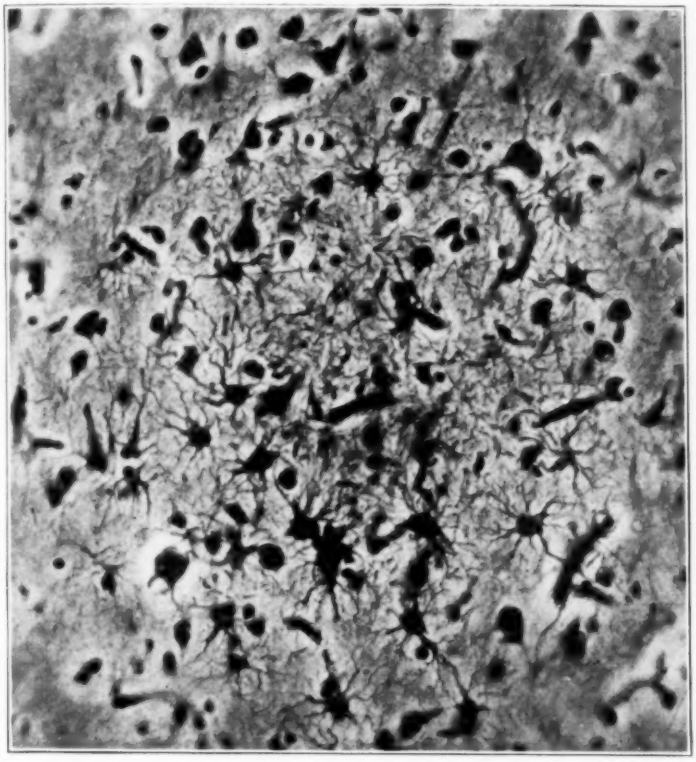

Fig. 16.—Perivascular gliosis, similar to that shown in figure 15. The closed vessel is surrounded by a small zone of degenerative fragmentation (Hortega's silver carbonate stain).

Second Operation.—In September, 1926, periarterial sympathectomy of the carotid and vertebral arteries was carried out by one of us (W. P.). Under ether anesthesia, an incision was made posterior to the upper end of the right sternocleidomastoid muscle and carried down to the great vessels. The superior cervical sympathetic ganglion was dissected out, but left intact with its proximal trunk and superior cardiac branch, while the upward going fibers on the carotid artery were cut and the adventitia of this artery was removed for a distance of from 2 to 3 cm. below its entrance into the skull.

A second perpendicular incision was then made over the lower end of the right sternocleidomastoid, and this muscle was cut and reflected. The stellate ganglion was isolated and likewise left intact, but its upward going fibers on the vertebral artery were cut. The accessory sympathetic ganglion which, in our experience,

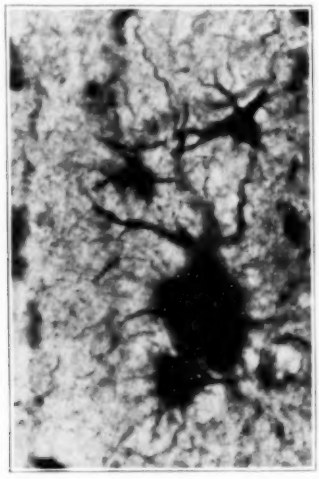

Fig. 17.—"Monster" glia cells (Cajal's gold chloride sublimate stain).

Fig. 18 (case 3).—Patient following the first operation.

has always been found fastened like a saddle about the vertebral artery was removed, together with the so-called vertebral nerve that passes from the upper end of this ganglion into the foramen riding on the artery. In order to make sure that no sympathetic fibers should escape, however, the artery itself was decorticated.

Technic.—As this operation had never been performed previously, so far as we are aware, a word as to the technic may not be out of place. By the use of deep, smooth, blunt retractors, an adequate exposure was obtained. The vertebral vein is perhaps the most annoying structure to displace, and caution should be used in its retraction. About 3 cm. of the vertebral artery was freed of adventitia with a small scalpel and a fine-pointed forceps. The artery cannot well be rotated as a whole, but the fibrous coat may be stripped back from either side.

Course.—The postoperative course was uneventful and the patient returned to his home in ten days. In the year following this right-sided sympathectomy of the arteries of the brain, the patient's general condition was excellent (fig. 18). Convulsive attacks continued, but with the following striking changes: At the onset of an attack, the left side of the face became white, while the right side became red. A few minutes later, the left side of the face also became red and the right eye somewhat "bloodshot." Whereas following the operation the right pupil was ordinarily smaller than the left, during attacks the two pupils were equally small. The convulsive movements after operation were strictly limited to the right side of the body, involving chiefly the right side of the face and the right arm.

In March, 1928, nineteen months after the operation, a letter was received from the boy's mother from which we may quote as follows: "Attacks begin with a bluish flush to face, turning head to right, jerking of right leg and a very loud patting with the right hand. Frequently he comes out of it singing. In normal health, attacks occur in the early morning only. A cold in January gave him seven attacks in forty-eight hours. He is well behaved and ambitious, eager to be useful, and he is allowed to help around his father's business. Thanking you sincerely for what you have been able to do for him."

The striking fact is that the unilateral sympathetic operation converted the convulsions into strictly unilateral attacks referable to the side of the brain not operated on, and that this strict unilaterality was still present at the time of the last observation recorded here—that is, nineteen months after operation.

A similar operation was carried out on the cerebral arteries of the left side in case 4. In this case, the whole brain was obviously involved in numerous multiple calcifications (fig. 4). However, the operation resulted in but little change in the child's condition, possibly because, in her case, the process had gone so far.

COMMENT

Calcification.—Various types of calcification have been reported in the literature. Rokitansky [2] described calcification of nerve tubes and nerve cells and vessels of the basal ganglions. Mallory [3] showed that the appearance of calcium in the vessels of the brain is preceded by the presence in them of colloid droplets. He pointed out later [4] that

2. Rokitansky, C.: Lehrbuch der pathologische Anatomie, Vienna, 1856.

3. Mallory, F.: A Contribution to the Study of Calcareous Concretions in the Brain, J. Path. & Bact. **3**:110, 1896.

4. Mallory, F.: Principles of Pathologic Histology, Philadelphia, W. B. Saunders Company, 1914.

hyalin regularly precedes calcification of vessels elsewhere and that in the central nervous system, in which it appears most often about the dentate nucleus of the cerebellum, this material appears as droplets in the vessel walls. The droplets may fuse to form a sheath and then calcify. This process takes place most frequently in the media but also in the adventitia. It affects the small arteries first and later the capillaries and the veins.

Hansemann [5] reported a case of calcification of vessels in the white matter and cited the report by Bamberger and Simon of two cases of idiots in whom calcification was observed. Apparently this was a diffuse process, also. Kaufman [6] stated that diffuse calcification is rarely seen in young persons, though "calcium metastasis" is a little more common. Bassoe and Hassin [7] recently reported a case of marked calcification in the cerebral white matter. They considered the primary pathologic change in their case as being in the capillary system and particularly in the tissues about the capillaries in which stasis had resulted in the deposition of calcium droplets.

A different type of process is the "calcium metastasis" that was described by Virchow [8] as being the result of an acute absorption of calcium from bone. It is seen in tuberculous caries, osteomalacia and like conditions. He pointed out that the sites elected for the deposit of such absorbed calcium were the stomach, lungs and kidneys. Tanaka [9] and Katase,[10] by increasing the intake of calcium experimentally, proved that calcium metastases may be caused by an increase in the calcium content of the blood. Küttner [11] suggested that a deficiency of carbon dioxide caused the calcium to be deposited locally, and Wells [12] pointed out that the stomach, the lungs and the kidneys (the sites of election) give up acid as a secretion and are, in consequence, more alkaline than other organs. Moreover, it is the arteries and not the

5. Hansemann, D.: Ein casuistischer Beitrag zur Verkalkung der Gehirngefässe, Verhandl. d. deutsche Path. Gesellsch. **2**:399, 1900.

6. Kaufman, E.: Lehrbuch der speziellen pathologische Anatomie, ed. 4, Berlin, 1907, p. 60.

7. Bassoe, P., and Hassin, G.: Calcification of the Cerebral Vessels with a Clinical Picture Simulating Brain Tumor, Arch. Neurol. & Psychiat. **6**:359 (Oct.) 1921.

8. Virchow, R.: Kalkmetastasen, Virchows Arch. f. path. Anat. **9**:619, 1956; Verkalkung der abgestorbenen Gehirnzellen, ibid. **50**:304, 1870.

9. Tanaka, M.: Ueber Kalkresorption und Verkalkung, Biochem, Ztschr. **35**:113, 1911.

10. Katase, A.: Experimentelle Verkalkung am gesunden Tiere, Beitr. z. path. Anat. u. z. aug. Path. **57**:516, 1914.

11. Küttner: Ein Fall von Kalkmetatase, Virchows Arch. f. path. Anat. **55**: 521, 1872.

12. Wells, H.: Metastatic Calcification, Arch. Int. Med. **15**:574 (Apr. 1) 1915; Chemical Pathology, ed. 4, Philadelphia, W. B. Saunders Company, 1920.

veins that tend to calcify, with the exception of the pulmonary veins (Butler [13]). This fact is additional evidence that a low tension of carbon dioxide favors this type of calcium deposition.

In the cases presented, evidence of calcification other than that in the brain was lacking, and in cases 3 and 4 the calcium content of the blood was studied and found to be normal. Further, it has already been pointed out that even if a generalized abnormal calcium metabolism had been present, the brain, if normal, would hardly have been singled out for deposition to the exclusion of the usual sites of election. It seems fair to conclude, therefore, that we were not dealing with cases of so-called metastatic calcification.

Wells [14] stated it as a rule that, except for the metastatic calcification of Virchow, degeneration of tissue always precedes pathologic calcification. As has been pointed out, the histologic picture seen in the brain in case 3 suggested strongly that the primary lesion was vascular. The pathologic process underlying this group of cases was probably a slowly progressive closure of the end arteries in the cerebrum, which produced an area of local necrosis, chiefly at the level at which the short pial vessel supply ends and the deeper blood supply begins. This area of degeneration then became secondarily calcified, and calcium was laid down in the walls of the involved small vessels.

This explains the typical and possibly pathognomic pictures seen in the roentgenograms. In the figures illustrating each of these cases, the larger deposits of calcium show hollow or umbilicated centers and, in some, stereoscopic examination shows the calcium arranged about the fissures of the cerebral cortex. The deposits, as we have pointed out, were located in general near the cerebral convexity and not in the brain stem. They were situated, therefore, in the zone of so-called terminal arteries farthest from the circle of Willis.

SUMMARY

Epilepsy in a father and all four of his children was associated in each case with changes in the brain leading to calcification of a peculiar type. In the father, convulsions did not occur until he was 48 years of age, although he had always suffered from migraine.

Each child showed normal mental development up to the age of 1 or 2 years; then each was mentally arrested. The first signs of epilepsy appeared respectively at 6 weeks, 5 months, 5 months and 2 days of age. The convulsions varied in form, including the individual extremities and face, or the whole musculature. One outstanding feature in all these attacks was the invariable accompaniment of pupil-

13. Butler, M.: Experimental Calcification in Mice, Proc. New York Path. Soc. **24**:79, 1924.

14. Wells (footnote 12, second reference).

lary change. In cases 1 and 4, the pupils were greatly contracted with each attack. In cases 2 and 3 the pupils were dilated at such times. Occasionally, when attacks were slight, only the pupillary spasm appeared, constituting a curious type of "petit mal," which the mother did not fail to observe, owing to her training as a nurse. The height of the convulsions was reached in one child at 14 months, in one at 4 years, at the time of death, and in one at 2 years; in the fourth (case 3), the attacks were variable up to the age of 8.

In two of the children there was a partial paralysis of one extremity, which gradually improved. Occasional swellings of the lymph glands were noted by the mother in three of the children. Severe convulsive attacks were induced in all by such intercurrent disturbances as diarrhea, tonsillitis and dentition.

In the four cases in which roentgenograms were obtained, discrete patches of calcification were found scattered through the brain, situated in general near the convexity of the cerebrum. Stereoscopic studies showed these patches to be umbilicated or in the form of furrows. This was evidently due to the fact that the calcification tended to take place at the junction of the cortical gray and white matter about a cortical fissure. Thus, the calcium about the fissure gave the impression of umbilication or furrow formation.

Operative excision in the only case in which the calcification was localized resulted in great improvement. Miscroscopic examination showed the process to be not parenchymatous, but primarily vascular. The glia changes were focused about the smallest blood vessels and the deposit of calcium had taken place about and in the walls of these small vessels. Areas of softening were not found, such as would result from closure of large arteries of supply. The term "endarteritis obliterans" or "endarteritis calcificans" is in one sense descriptive. The nature of the process that caused the terminal vessels to close and calcify, however, is not clear. An infectious process in a family group seems unlikely, though not impossible. The suggestion must also be entertained that recurring obliterative vasomotor spasm might account for both the convulsions and the closure of the small vessels. In any case, the calcification was undoubtedly secondary to the localized chronic destruction of tissue due to closure of the small vessels.

A new operation is described, periarterial sympathectomy of the carotid and vertebral arteries of one side. This resulted in the conversion of bilateral convulsions into unilateral convulsions. This result, together with the constant appearance of pupillary change during each attack, might speak for vasomotor spasm as the cause both of the convulsions and of the changes in the end arteries. Why the pupillary change should be dilatation in two cases and contraction in two is hard to explain.

The appearance of the same condition in a father and four children, together with the peculiar and constant manifestations in each case, would seem to justify the belief that we are dealing with an independent disease process. The outstanding clinical features are the convulsions and the calcification, although both these factors are secondary manifestations of the primary process. "Cerebral calcification epilepsy" would therefore describe the obvious manifestations of the disease. The actual pathologic process involved, however, is more clearly indicated by "endarteritis calcificans cerebri."

TUMORS OF THE BRAIN WITH ACUTE ONSET AND RAPIDLY PROGRESSIVE COURSE

"ACUTE BRAIN TUMOR" *

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The symptoms of intracranial tumor may appear suddenly and may advance rapidly, due either to bleeding into a growth, to the precipitate occurrence of obstructive hydrocephalus, swelling or edema, or to interference with the blood supply of a part of the brain. The onset of symptoms may be sudden as the result of hemorrhage into a tumor in a relatively "silent" area of the brain. Acute dilatation of the ventricular system is not rare when a growth, especially one in the posterior cranial fossa, begins to cause pressure on some part of the ventricular channels. "Swelling" of the brain may come on quickly in infiltrating tumors. Growths, especially those situated in or near a temporal lobe, not infrequently interfere with the circulation of blood in a middle cerebral artery or one of its main branches, the result of which is a large area of encephalomalacia with the sudden appearance of marked symptoms.[1]

In the majority of instances of this kind in which acute symptoms occur, the patients have suffered for a preceding period from disturbances later recognized as symptoms due to the intracranial lesion.

In the thirty-seven cases that form the basis for this paper, all of which were verified by operation or autopsy, the patients did not present a history of intracranial disturbances until the actual beginning of the illness for which they were admitted to the hospital. The onset was usually sudden and the advance of the symptoms unusually rapid, so that often the clinical picture was more like that of an acute disease than that of an intracranial neoplasm.

We have considered the cases only from the standpoint of the beginning and the progression of the symptoms and have, in the main,

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* From the Neurological and Surgical Services and the Laboratories of Mount Sinai Hospital.

1. Elsberg, C. A., and Riley, H. A.: Differential Diagnosis Between Cerebral Degeneration, Infiltrating Cerebral Neoplasm and Infiltrating Cerebral Neoplasm with Degeneration, Arch. Neurol. & Psychiat. **15**:48 (Jan.) 1926.

left out of consideration the more or less speculative question of the actual duration of the tumor itself, although histologically the wealth of cellular elements and proliferation of cells was an evidence of active growth.

The researches of recent years, especially those of Bailey and Cushing [2] and of Strauss, and of one of us (J. H. G),[3] have demonstrated that primary malignant tumors of the central nervous system are not rare and that the most malignant and rapidly growing type that occurs is that to which the latter authors gave the name of spongioblastoma multiforme. Strauss and one of us (J. H. G.)[4] referred to the rapid growth and the swift advance of symptoms in this variety of neoplasm. In their monograph on tumors of the glioma group, Bailey and Cushing stated that from the earliest symptoms the average period of survival of patients with spongioblastoma multiforme of the brain was thirteen months, and that if the patients were not subjected to operative intervention the average duration of life was only three months.[5]

Besides the publications of these writers, the literature on the subject contains only isolated reports of cases.

Inkster [6] published a report of a "case of intracranial neoplasm with unusually rapid development of symptoms." A man, aged 38, had rapidly advancing hemiplegia with few other symptoms. He succumbed twenty-four days after the first evidence of illness, and at the postmortem examination was found to have a "chocolate colored tumor," a "gliosarcoma," in the left cerebral hemisphere. The author did not give further details of the histologic nature of the growth, but it seems probable that this was really a spongioblastoma.

A number of cases have been reported because the onset and course of the disease was difficult to distinguish from acute encephalitis. For example, Rabinowitsch [7] published the report of a case in a man, aged 22, who died five months after the onset of the disease. The main symptoms were violent headaches, drowsiness progressing to stupor, rigidity of the neck, anisocoria and incontinence. Autopsy revealed a "gliosarcoma" of the left optic thalamus and third ventricle.

2. Bailey, P., and Cushing, H.: The Tumors of the Glioma Group, J. B. Lippincott Company, Philadelphia, 1926.

3. Globus, J. H., and Strauss, I.: Spongioblastoma Multiforme, Arch. Neurol. & Psychiat. **14**:139 (Aug.) 1925.

4. Globus, J. H., and Strauss, I.: Footnote 3 (p. 190).

5. Bailey, P., and Cushing, H.: Footnote 2 (pp. 108 and 114).

6. Inkster, J.: A Case of Intracranial Neoplasm with Unusually Rapid Development of Symptoms, Lancet **2**:1074, 1927.

7. Rabinowitsch, A.: Ein Fall von Hirntumor unter dem Bilde einer epidemischen Encephalitis, Deutsche Ztschr. f. Nervenh. **88**:67, 1925.

Reports of other cases of this kind have been published, and without doubt every neurologist has had under his care and observation patients with similar symptoms.

REPORT OF CASES

Synopses of the clinical records of some of our patients are given.

CASE 1.—*History.*—Fannie K., aged 51, was admitted to the hospital and died, July 5, 1922. Eight weeks before admission she was said to have had an attack of polyarticular rheumatism with fever. Four weeks later, while sitting up in bed, she had a sudden attack of dizziness so that she fell out of bed and fractured the nasal bones. She recovered. Three weeks later, she suddenly had a violent throbbing headache in the right frontal and temporal regions with vomiting and drowsiness. The following morning, she had a complete left facial paralysis, weakness of the left upper extremity and incontinence.

Examination.—On admission, she was in deep stupor. The pupils were unequal, the right larger than the left; there was weakness of the left side of the face and of the left upper and lower extremities, with a bilateral Babinski sign. Some rigidity of the neck and a distinct Kernig sign were present.

Course.—She became rapidly worse, and died in coma less than twenty-four hours after admission.

Autopsy.—On examination, the brain was somewhat flattened. The vessels were markedly thickened. The right temporal lobe was soft and fluctuating and on incision was found to be replaced by a large, soft, brownish-red neoplastic mass. Recent hemorrhages were seen in the mass. The lateral ventricles and the basal ganglia were deflected to the left by the tumor.

The tumor, a spongioblastoma multiforme, presented a fairly uniform histologic picture. The dominating type of cell was large, irregular in shape, contained one or more nuclei, and in some instances gave off blunt processes (fig. 1). Fusiform and flasklike cells were also found in large numbers. The tumor tissue was marked by extensive areas of necrosis, numerous hemorrhagic foci and great vascularity.

CASE 2.—*History.*—G. L., a man, aged 52, was admitted to the hospital on June 6 and died on June 9, 1923. He had been deaf since childhood. Three weeks before admission he began to suffer from severe frontal headache, especially on the right side, and this persisted with undiminished severity.

Examination.—The man was mentally dull and noncooperative. The pupils were unequal, the left larger than the right; the left pupil reacted poorly to light. Some ptosis on the left, and distinct facial weakness on the lower left were present, and the tongue, when protruded, deviated slightly to the left. The margins of the disks were slightly blurred. There was no weakness of any of the limbs. The tendon reflexes were hyperactive but equal on the two sides. The abdominal reflexes were weak; the Babinski, Gordon and Oppenheim signs were present on the right. Some hypalgesia and diminution of vibratory and position sense were present in the left lower extremity.

Course.—On June 7, marked rigidity of the neck was present with a Kernig sign. Lumbar puncture yielded a clear fluid with 8 cells; the globulin was not increased; the Wassermann test was negative. The patient became deeply comatose and died on the third day after admission.

Autopsy.—The dura was tense and bulging. There was a moderate degree of congestion in the smaller pial vessels over the entire dorsolateral surface of the

cerebral cortex and in the interpeduncular space. The gyri of the cerebral hemisphere presented a marked degree of flattening.

In the frontoparietal region of the right cerebral hemisphere, there was a neoplastic mass. It measured about 5 cm. in its long diameter and was surrounded by a wide zone of edema; another independent mass was found more anteriorly on the right side of the corpus callosum. In the midbrain, marked congestion with areas of softening was noted (fig. 2).

Section through the center of the tumor displayed a densely cellular picture. The most conspicuous type of cell was a large mononuclear cell (fig. 3), irregular in outline and occasionally giving off a long process directed to the wall of a blood vessel. Giant cells with their nuclei, two to six in number, arranged most com-

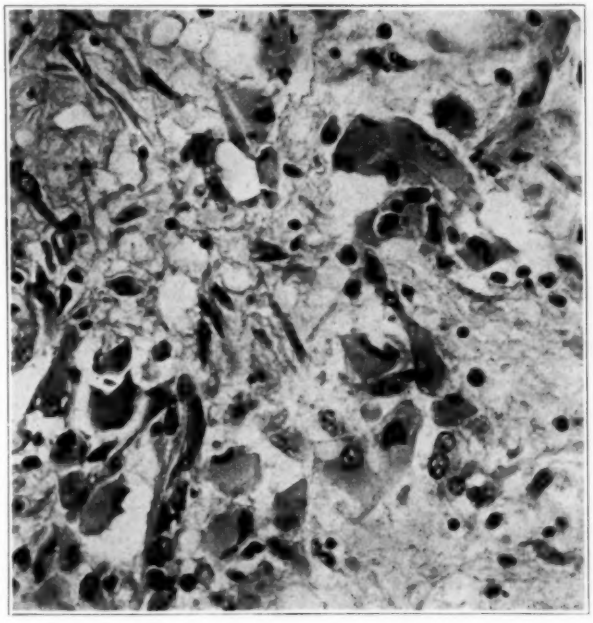

Fig. 1 (case 1).—Cell type of the tumor—a spongioblastoma multiforme. Hematoxylin stain; × 300.

monly at the periphery of the cell, were also frequently seen. The tumor was a typical spongioblastoma.

Case 3.—*History.*—Samuel K., a man, aged 50, was admitted to the hospital, Jan. 22 and died, Feb. 16, 1926. Three months before, he began to suffer from headache, with increasing weakness of the right upper and lower extremities. After a few weeks, he became irrational, lethargic, and had increasing difficulty with speech. He was referred to the hospital as having epidemic encephalitis.

Examination.—On admission he was drowsy, so that examination was not satisfactory. There seemed to be marked motor and sensory aphasia. A right hemiparesis with the tendon reflexes more active on the right and a right inexhaustible ankle clonus were present. The abdominal and cremasteric reflexes were diminished on the right.

The fundi showed a low grade of papilledema. The pupils were equal and reacted promptly to light and in accommodation. There was a right facial paralysis, supranuclear in type. On lumbar puncture, the spinal fluid was under increased pressure, was distinctly xanthochromic and contained 12 lymphocytes to the cubic millimeter.

Course.—The aphasia became more marked and the left pupil distinctly smaller than the right. The papilledema became more marked with hemorrhages, and the hemiparesis became complete with bilateral ankle clonus and Babinski signs. An exploratory operation (Dr. Neuhof) revealed a tumor deep in the left parieto-temporal region, which could not be removed. A large subtemporal decompression was performed. Death occurred four days after the operation.

Fig. 2 (case 2).—Gross appearance and location of the two separate tumor masses.

Autopsy.—Only a partial autopsy was performed. Through the existing surgical incision, only the left hemisphere was removed. The surface of the brain showed evidence of increased intracranial pressure. The pia-arachnoid over the frontal lobe was thickened and cloudy. A large tumor mass lay buried within the sylvian fissure. It was infiltrating in character, extended deeply into the basal ganglia and came to the surface in the region of the insula. It was vascular and granular in appearance (fig. 4).

The histologic features were those of spongioblastoma multiforme (fig. 5) and included the giant spongioblasts and high vascularity alternating with wide zones of necrosis.

Case 4.—*History.*—Morris G., a man, aged 54, was admitted to the hospital, July 20 and died Aug. 11, 1926. He had had influenza in the winter of 1925-1926. There had been two weeks of headache and dizziness. He became drowsy, so that

he would drop asleep whenever he sat down. The symptoms became more marked and for one day he had been in stupor.

Examination.—The patient was in a stupor; the fundi were normal; the right pupil was larger than the left, but both reacted well to light. Movements of the eyeballs could not be tested. There was some tremor of both hands. The power of the limbs could not be tested; the tendon reflexes were equal on the two sides, but a suspicion of a Babinski sign was present on the left. A roentgen examination

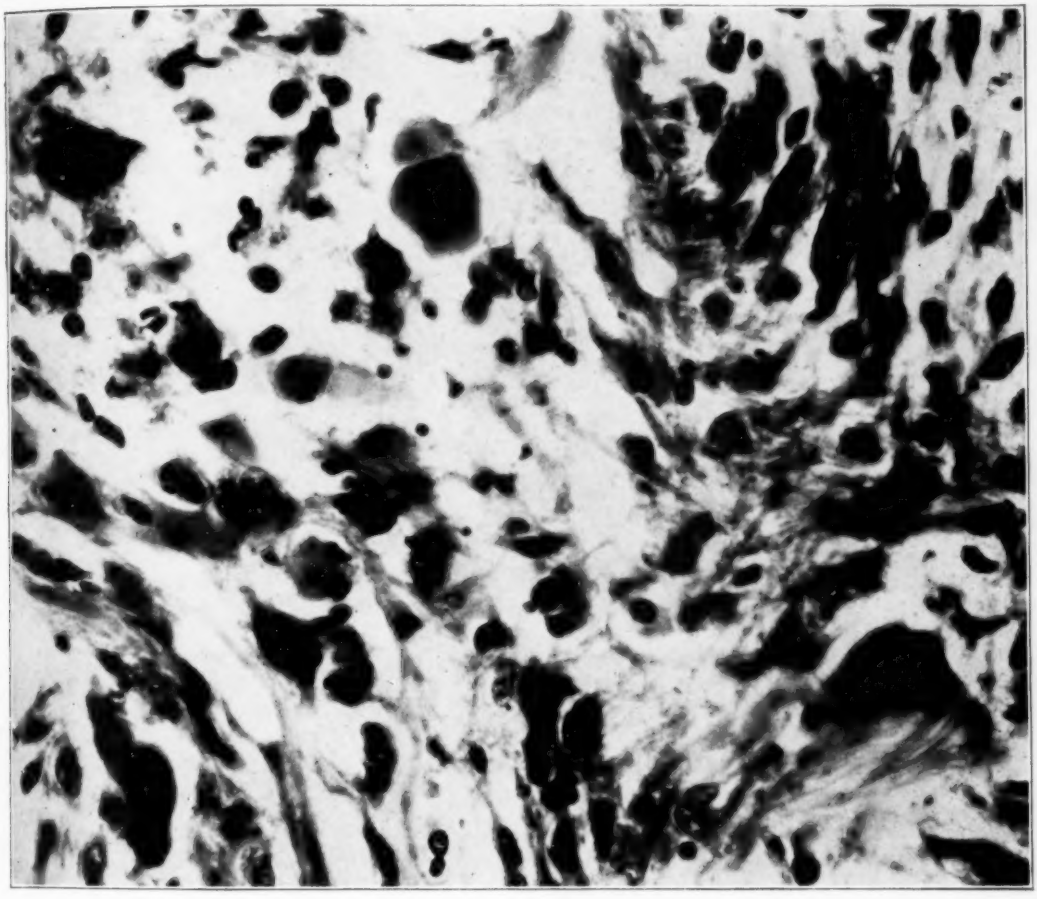

Fig. 3 (case 2).—Numerous multinuclear cells, among which there are many giant cells. Hematoxylin; × 550.

gave negative results. Lumbar puncture showed: a clear fluid, escaping under increased pressure; 4 cells; globulin, ++; the Wassermann test was negative.

Course.—The patient was believed to be suffering from encephalitis and received intravenous dextrose therapy. He remained in about the same condition until the beginning of August. Then the stupor became deeper; examination of the fundus revealed a beginning papilledema, the tendon reflexes on the left side became more active than those on the right, and a definite Babinski sign was now present on the

left. The diagnosis was now changed to that of tumor and a right exploratory craniotomy was performed. After the bone flap had been turned down the dura was not found tense, the brain pressure was little increased and evidence of a tumor could not be discovered. A subtemporal decompression was left at the base of the bone flap.

The patient did not improve; he remained in stupor and died, Aug. 11, 1926.

Autopsy.—The brain showed evidence of intracranial tension. A large tumor occupied the right temporo-occipital lobe. It was well demarcated from adjacent tissue and was easily enucleated (fig. 6).

Histologic examination revealed a densely cellular neoplasm with extensive areas of degeneration. There were numerous areas of roset formation made up of large,

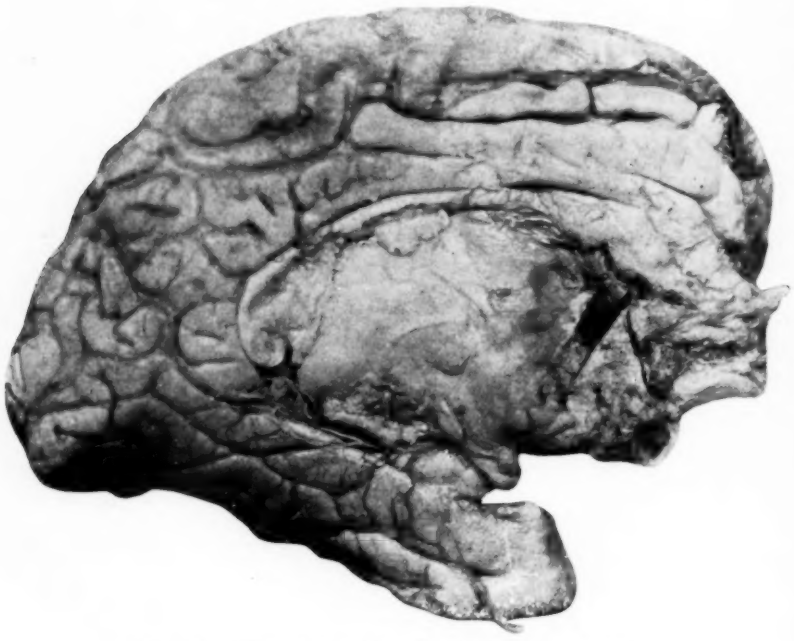

Fig. 4 (case 3).—Location and appearance of the tumor.

irregular, multinuclear giant cells. Occasional mitotic figures could be seen. The microscopic picture was typical of a rapidly growing malignant tumor of the brain, spongioblastoma multiforme (fig. 7).

CASE 5.—*History.*—George B., a man, aged 56, was admitted to the hospital, Dec. 9 and died Dec. 29, 1926. One year before, he fell a distance of 35 feet, injuring his head and right leg, but recovered entirely. Five weeks before admission, he had sudden severe headache followed by dizziness and blurring of vision, poor memory and some disturbance in speech, all of which had steadily grown worse.

Examination.—The patient was much emaciated and in poor general condition. He was garrulous and somewhat euphoric, and his speech was slow and drawling. His vision was poor. There was a left homonymous hemianopia. The fundi showed a marked bilateral papilledema. The pupils were unequal, the left larger

than the right, and both reacted sluggishly to light and in accommodation. The left cornea was anesthetic and the right corneal reflex diminished. The left palpebral fissure was larger than the right, and there was a distinct weakness of the left side of the face, supranuclear in type.

The tendon reflexes were not hyperactive and were equal on the two sides. Coarse tremor and pseudo-athetoid movements were present in the fingers of the left hand. Marked disturbances of position sense were present in the left upper and lower limbs.

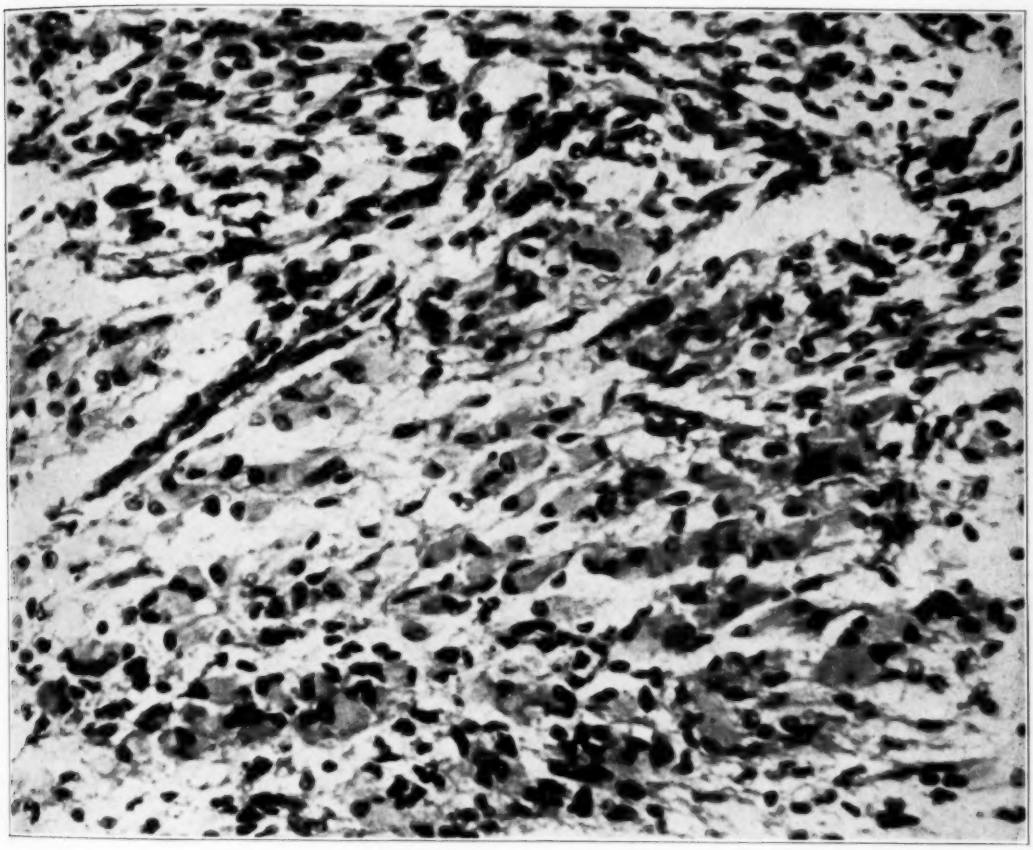

Fig. 5 (case 3).—The cell type in this tumor is similar to that in case 8 as shown in figures 16 and 17; with the only difference that the cells here are much smaller. Hematoxylin; × 300.

Lumbar puncture showed a normal spinal fluid; air, injected by the lumbar route, showed a deformity of the body of the right ventricle and a dislocation of the ventricles toward the left. The patient was treated with hypertonic dextrose solution, as he was not in a condition for major surgical intervention. By ventricular puncture, only 15 cc. of fluid could be removed.

Course.—By December 21, the condition was better so that an exploration under local anesthesia seemed permissible. At the operation (performed by one of

us, C. A. E.), a tumor was located 3 or 4 cm. below the surface of the temporal lobe. The lobe was incised, but the growth was so extensive that its removal was not attempted. Specimens were removed for verification, and the bone flap was replaced after bone had been rongeured away at its base for decompressive purposes.

The patient died eight days after the operation.

Autopsy.—Postmortem examination showed the following: The brain was under increased intracranial pressure. The basal surface of the right temporo-occipital lobes was adherent to the underlying skull. On the lateral surface of the temporal lobe in the region of the middle half of the superior temporal convolution, there was a crater-like opening leading to a large area filled with sanguineous necrotic material. This area occupied about the posterior half of the

Fig. 6 (case 4).—Gross appearance and location of the tumor.

temporal lobe and the adjacent part of the occipital lobe. On sectioning, a large granular, graying mass was found. It was oval in shape and occupied almost the entire width of the occipital lobe, extending downward to the base of that lobe and upward to about the level of the sylvian fissure (fig. 8).

Histologically, the tumor consisted of densely cellular, undifferentiated glia cells, among which were many large multinucleated spongioblasts (fig. 9).

CASE 6.—*History.*—Louis G., a man, aged 48, was admitted to the hospital on March 17 and died, April 15, 1927. Four months before admission, he had a large furuncle on the back of his neck which was incised and drained. Nine weeks before, he had an attack of dizziness and headache which lasted for a week. One week later, the left upper and lower limbs suddenly became weak so that he fell down while attempting to stand. The weakness persisted unchanged until one

week before admission. Then severe headache recurred and the loss of power became more marked. On the following day he became drowsy, and incontinent of urine and feces. All of the symptoms had grown worse.

Examination.—On admission, the patient was in a stupor and was difficult to arouse. Examination was unsatisfactory. The fundi showed a blurring of the disk margins. There was slight weakness of the left external rectus and a central facial weakness on the same side. The tongue deviated to the left when protruded.

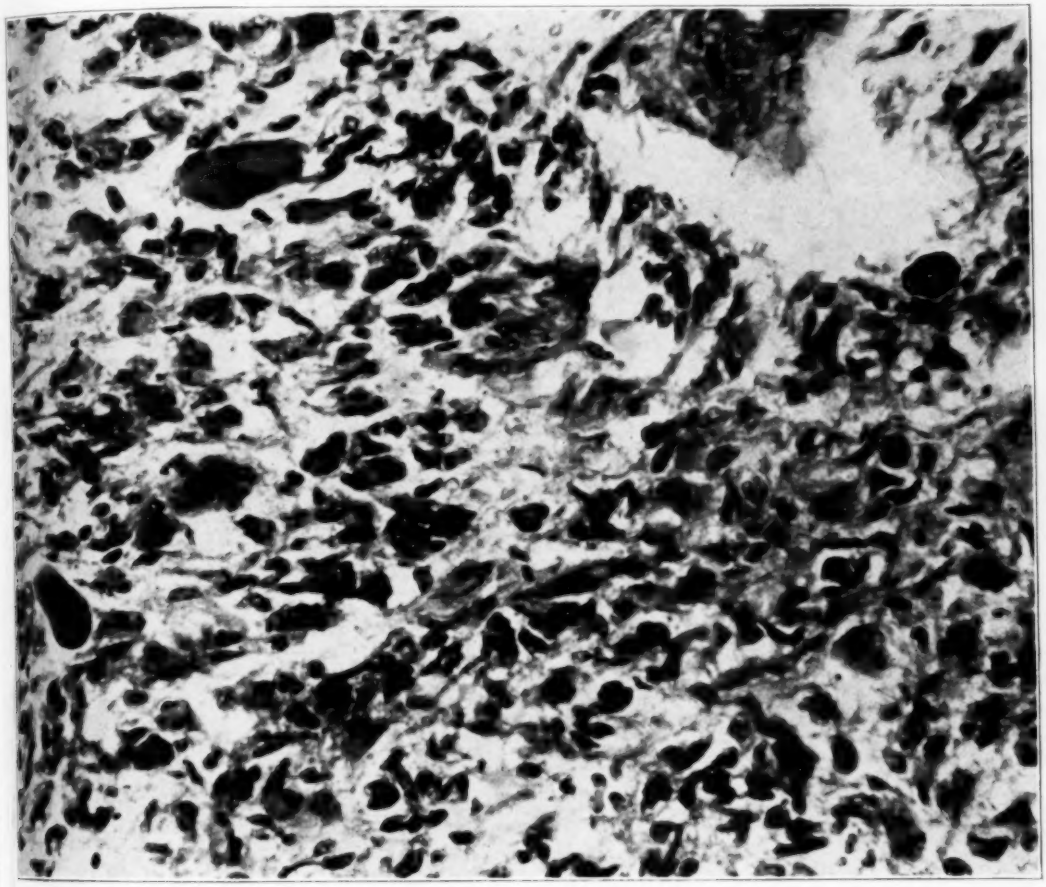

Fig. 7 (case 4).—The densely cellular structure of the tumor and the character and number of the giant cells. Hematoxylin; × 300.

There was a left hemiparesis with increase of tendon reflexes and a Babinski sign. The abdominal reflexes could not be elicited. Roentgen examination gave negative results. The spinal fluid escaped through the needle under markedly increased pressure and contained 100 cells to the cubic millimeter, 90 per cent of which were lymphocytes.

Course.—On March 25, the patient was somewhat brighter and more cooperative. Roentgen examination of the skull after the injection of air by the lumbar route showed a filling defect of the anterior part of the right lateral ventricle.

There was a gradual advance in the changes in the fundi and on April 2 the examination revealed a distinct papilledema on both sides. The hemiparesis was much less marked.

On April 8, craniotomy was performed under local anesthesia (by one of us, C. A. E.). A large osteoplastic flap was turned down on the right side. The tension of the dura was not increased. On incision of the dura, the brain was

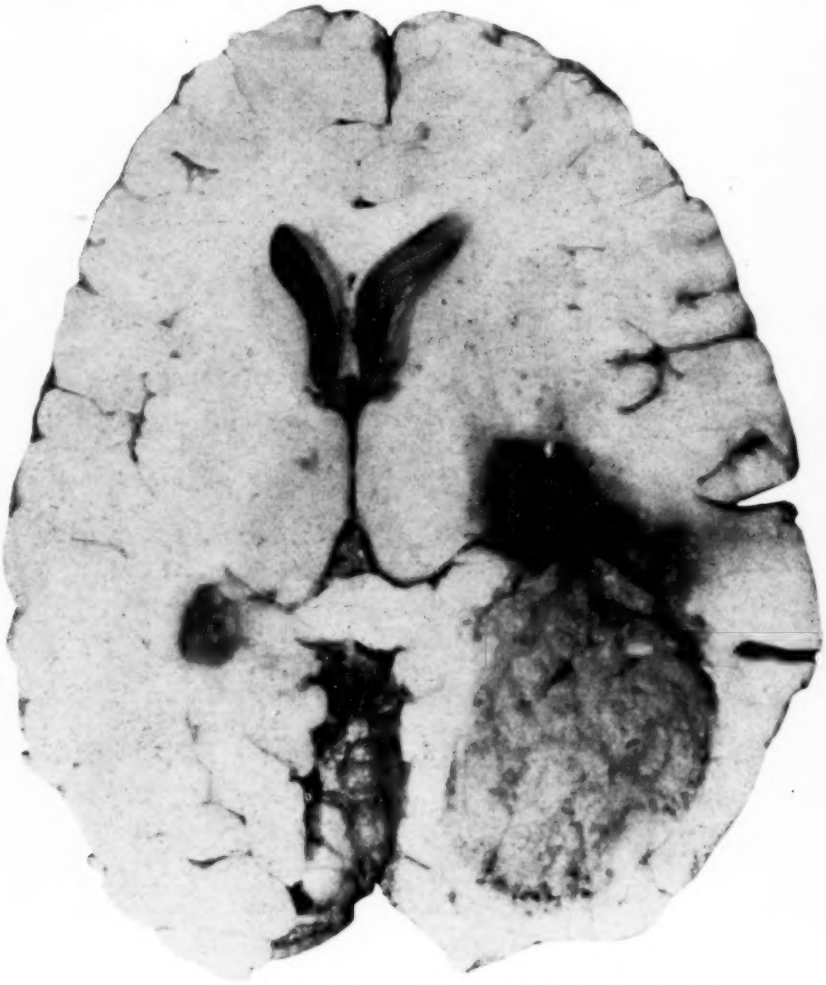

Fig. 8 (case 5).—Gross appearance and location of the tumor.

not under increased pressure and did not bulge through the opening of the dura. Exploration gave entirely negative results except for a vague resistance felt deep in the temporal lobe with the exploring needle. A large subtemporal decompression was left.

The condition was not improved by the operation. The patient became more and more drowsy and stuporous, and died one week later.

Autopsy.—The brain was voluminous. The right hemisphere was somewhat larger than the left. The right frontal lobe was soft and somewhat flattened; the right temporal lobe in its anterior half was hard and nodular. This was particularly true on the inferior surface in the region of the uncus. The vessels of the base of the brain did not show any changes except that the middle cerebral artery was compressed a short distance from its origin and almost totally occluded at that point of its course, in the sylvian fissure, where it was surrounded by the invading

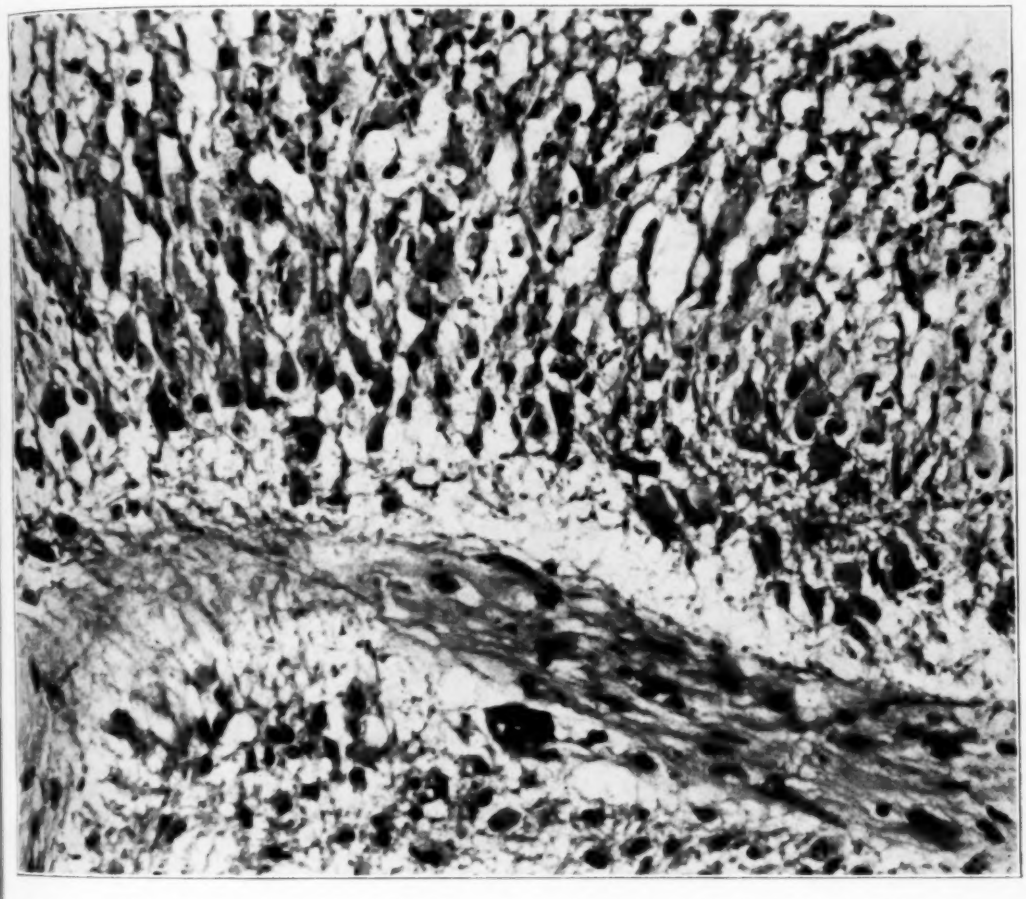

Fig. 9 (case 5).—The radial cell arrangement. The cell type is the same as in cases 2, 3, 7 and 8 (figs. 3, 5, 14, 16 and 17). Hematoxylin; × 300.

tumor mass. On horizontal section of the brain, the tumor mass was found invading the right lenticular nucleus, the island of Reil and the adjacent portion of the temporal lobe (fig. 10). In its extension downward, the tumor invaded almost the entire half of the temporal lobe. An area of softening was also seen in the frontoparietal region, on a level with the under surface of the corpus callosum. The area of softening (fig. 11) extended into the white substance adjacent to the island of Reil. This was undoubtedly caused by the occlusion of the vessel.

Sections of the tumor revealed a densely cellular, vascular new growth with numerous areas of degeneration and hemorrhage. There were many large, irregular cells with eccentric nuclei, as well as numerous multinuclear giant cells (fig. 12). Mitotic figures were frequently encountered. The picture was fairly typical of spongioblastoma multiforme.

CASE 7.—*History.*—Betty C., a woman, aged 53, was admitted to the hospital on April 26 and died, April 29, 1927. Hysterectomy was performed for sarcoma

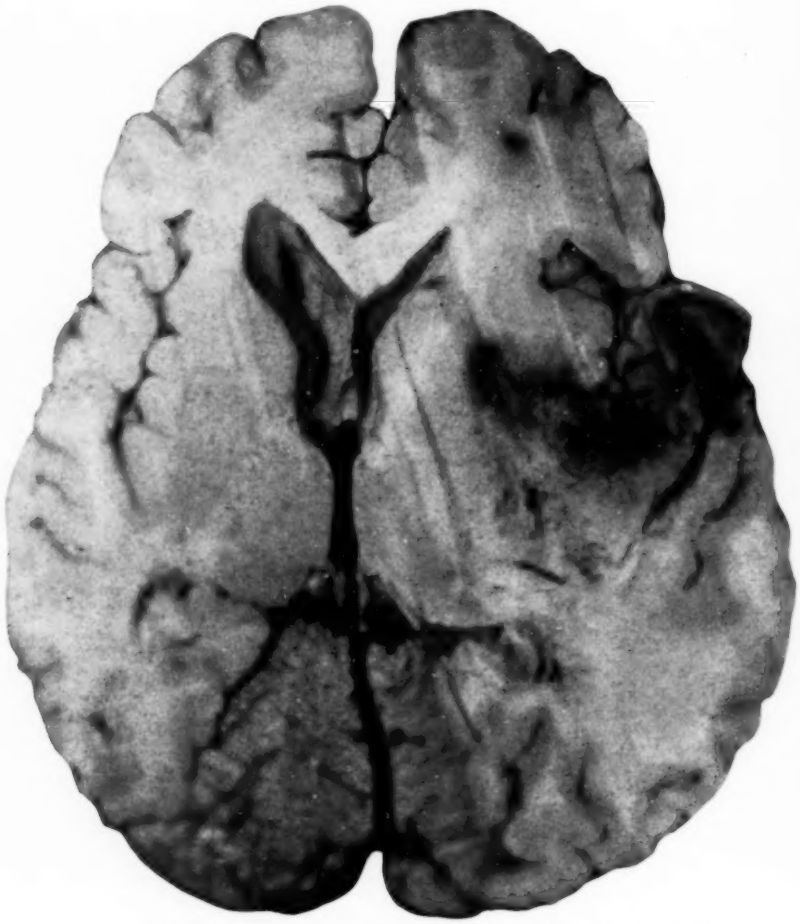

Fig. 10 (case 6).—Gross appearance and location of the tumor.

of the uterus ten years before. Eight weeks before admission she began to suffer from headache and developed a gradually increasing disturbance in speech. She was under treatment in another hospital, from which she was discharged with the diagnosis of cerebral arteriosclerosis. The symptoms steadily grew worse; two days before, she became drowsy, and for a day before admission she had been in stupor.

Examination.—The patient could be aroused only with great difficulty. The fundi showed a bilateral papilledema of 1.5 diopters with considerable neuroretinitis. The pupils were equal and reacted well to light and in accommodation. The left cornea was anesthetic. Weakness of the right side of the face and deviation of the tongue to the left when protruded was present. The right upper

Fig. 11 (case 6).—Section of brain with area of softening.

extremity was spastic; the right lower limb was flaccid. The tendon reflexes in the lower extremities were hyperactive, more on the left than on the right; bilateral Babinski confirmatory signs were present.

The urine contained 3.5 per cent of sugar, with acetone, and the blood sugar was 1.3.

Course.—The patient remained in deep stupor, and died on the third day after admission to the hospital; she was considered to have succumbed as the result of a cerebral neoplasm into which a hemorrhage had occurred.

Autopsy.—At the temporal pole of the left hemisphere there was an irregularity in the contour of the surface of the brain. This area presented an increased resistance to the palpating finger, while in its immediate vicinity the brain substance was softened. On section a neoplasm in the inferior temporal gyrus of the left

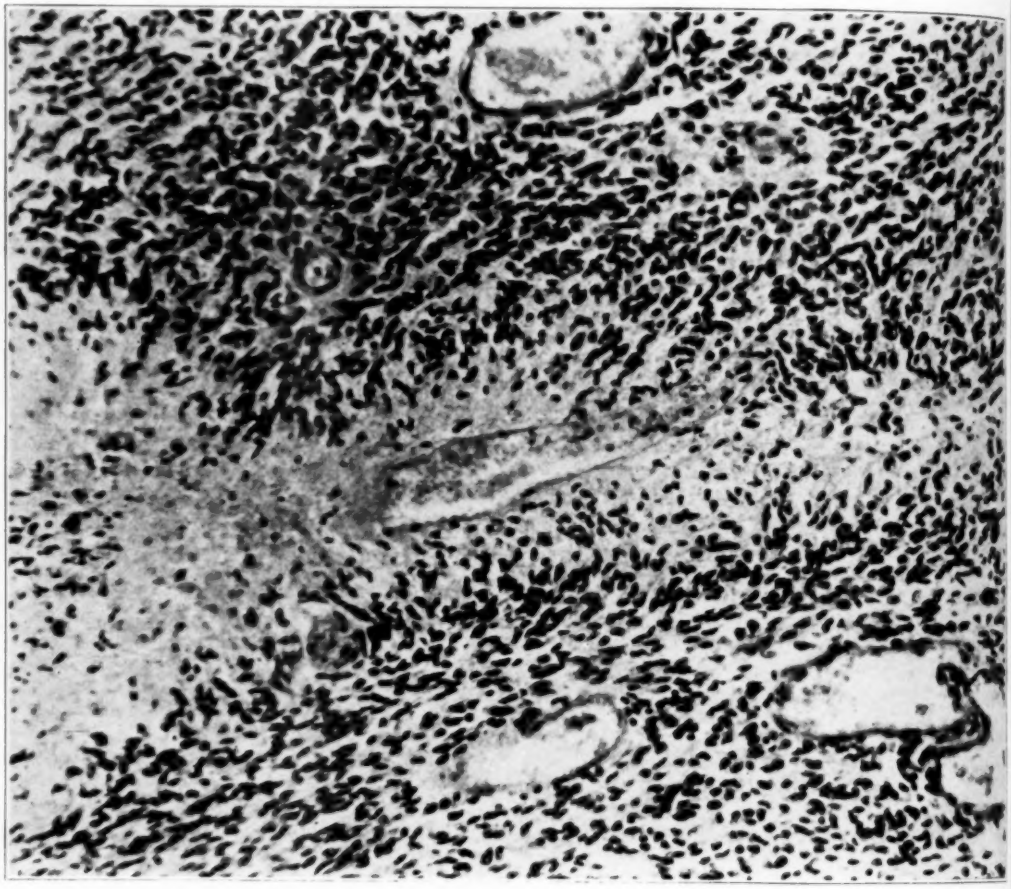

Fig. 12 (case 6).—Average field under low magnification, illustrating the radial cell arrangement and the cellular density of the growth. Hematoxylin; × 225.

hemisphere was found; it extended backward as far as the anterior half of the occipital lobe. It measured about 5 cm. in transverse diameter; it was fleshy and hemorrhagic (fig. 13).

Histologically, the tumor was densely cellular and surrounded by a wide zone of softening. The cell structure and arrangement were typical of spongioblastoma multiforme (fig. 14).

CASE 8.—*History.*—A. C., a man, aged 42, was admitted to the hospital on June 29 and died, July 5, 1927. Three months before, he suddenly began to suffer from severe headache, vomiting and fever, which was diagnosed as "grip." After one week, he complained of feeling dizzy; his vision became blurred; he was at times disoriented and complained of pain in the right lower extremity and of weakness in the right upper limb. Headache and vomiting continued, and he began to have difficulty in speech. Two days before admission, he became drowsy and incontinent of urine and feces.

Examination.—Examination was unsatisfactory on account of lack of cooperation. There seemed to be aphasia and apraxia. Bilateral pyramidal tract signs were present and were more marked on the right. A bilateral Babinski sign was present and more marked on the right; the abdominal reflexes could not be elicited. The fundi showed a bilateral papilledema of 2 diopters with exudates and hemorrhages. The right pupil was smaller than the left; the left pupil reacted

Fig. 13 (case 7).—Gross appearance and location of the tumor.

sluggishly and the right did not react to light or in accommodation. There was distinct flattening of the right side of the face.

Course.—On July 3, both eyes deviated to the left; there was occasional twitching of the right upper limb; a bilateral Kernig sign was present. The patient was in poor condition, with irregular fever, rapid pulse, cyanosis and stupor.

Death occurred on July 5, six days after admission.

Autopsy.—The brain was voluminous; its surface showed evidence of increased intracranial tension; the left hemisphere was somewhat larger than the right. On section a large tumor mass was found in the left occipital lobe, mesial to the posterior horn, and spreading into the pulvinar (fig. 15).

Histologically, the neoplasm was an undifferentiated gliogenous growth of the character frequently seen in association with tuberous sclerosis. The cells were numerous. They were large and some of them were multinuclear. They were of the type and arrangement seen in spongioblastoma (figs. 16 and 17).

CASE 9.—*History.*—Helen B., a woman, aged 43, was admitted to the hospital on Jan. 14 and died, Feb. 23, 1928. Headaches and twitching of the muscles of

the right side of the face and right upper extremity were of two or three months' duration; a sensation of numbness and paresthesias in and weakness of the right upper limb had existed for one month. Sixteen hours before admission, severe convulsive twitchings of the muscles of the face and arm on the right side occurred, with difficulty of speech.

Examination.—The patient was in good condition and cooperated well. Speech was dysarthric. The optic disks were normal, although somewhat pale. The right

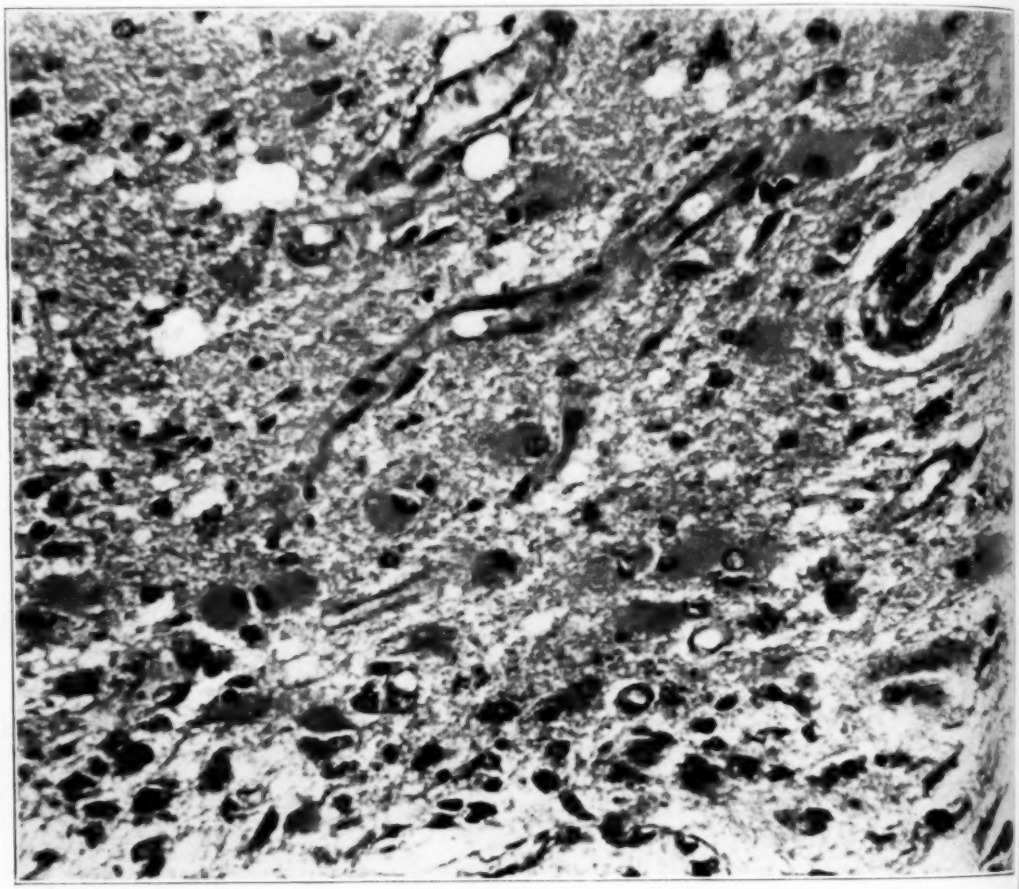

Fig. 14 (case 7).—Spongioblastoma multiforme. The cells are of the same type as in cases 2, 3, 5 and 8, but they are not as densely packed (compare with figs. 3, 5, 9, 16 and 17). Hematoxylin; × 300.

pupil was larger than the left; both pupils reacted sluggishly to light but promptly in accommodation. Movements of the eyeballs were good in all directions. Right facial weakness, involving especially the lower branches of the nerve, was present. The deep reflexes were more active on the right, and there was a Babinski sign on the same side. Astereognosis and diminution of all forms of sensation were present in the right hand.

On lumbar puncture, the fluid was clear and pressure was not increased; there were three cells; the globulin test was negative. Roentgen examination of the skull gave negative results.

Vaginal examination revealed an enlarged and eroded cervix, and malignant disease was suspected. On this account, a curettage was done and a specimen of the cervix removed, but the laboratory report was negative for malignant disease.

Course.—While under observation, the patient had several jacksonian seizures which always involved the muscles of the face and the upper extremity on the right side.

Fig. 15 (case 8).—Gross appearance and location of the tumor.

On February 17, craniotomy (Dr. Ira Cohen) was performed under local anesthesia. A large osteoplastic flap was turned down and the dura incised. In the cortex of the left parietal lobe was a brownish tumor, not well demarcated from the surrounding normal brain tissue. A tumor mass, 5 by 3 by 2 cm., was excised. Its gross appearance was that of a glioma.

After the operation, the paralysis of the right arm and the right side of the face was complete. The condition was satisfactory for three days. The patient then became stuporous, the pulse rapid and feeble, and death occurred one week after the operation.

Autopsy.—The left cerebral hemisphere presented a large defect in the brain substance in the area corresponding to the parietal lobe which was the site of the operative area. This shallow cavity was surrounded by a narrow zone of necrotic tissue which invaded the brain substance and extended forward into the frontal lobe and backward into the occipital lobe. There were several areas scattered over the cerebral cortex which presented an unusual rubbery hardness to the touch. On section, such an area presented an indistinct demarcation between gray and

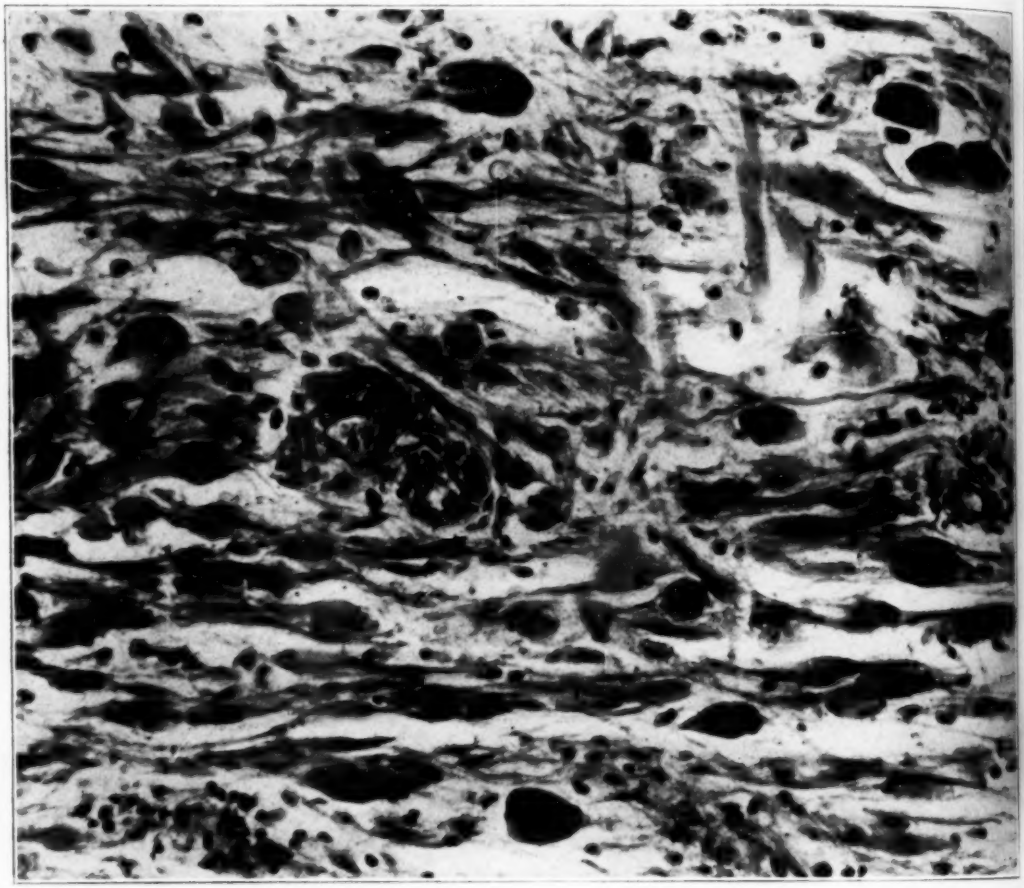

Fig. 16 (case 8).—Section from the center of the tumor mass, illustrating the uniform character of its cellular structure. Hematoxylin; × 230.

white substance, suggesting the possible existence of tuberous sclerosis. The vessels at the base of the brain were normal.

The histologic structure of the tumor was that of spongioblastoma multiforme (fig. 18).

CASE 10.—*History.*—Michael P., a man, aged 46, was admitted to the hospital on April 12 and died, April 27, 1921. For two years, the patient had had attacks of precordial pain which was diagnosed as angina pectoris. Three weeks before

admission, he began to suffer from severe headache over the occipital and frontal regions and began to lose interest in his surroundings. He became drowsy and apathetic, vomited frequently and at times complained of diplopia. During the patient's stay at another hospital, all of the symptoms became intensified, and he was admitted in stupor.

Examination.—There was marked tenderness on percussion over the left parietal region. Bilateral papilledema, of from 2 to 3 diopters, with hemorrhages, was present. The pupils were unequal, and reacted sluggishly to light; there was

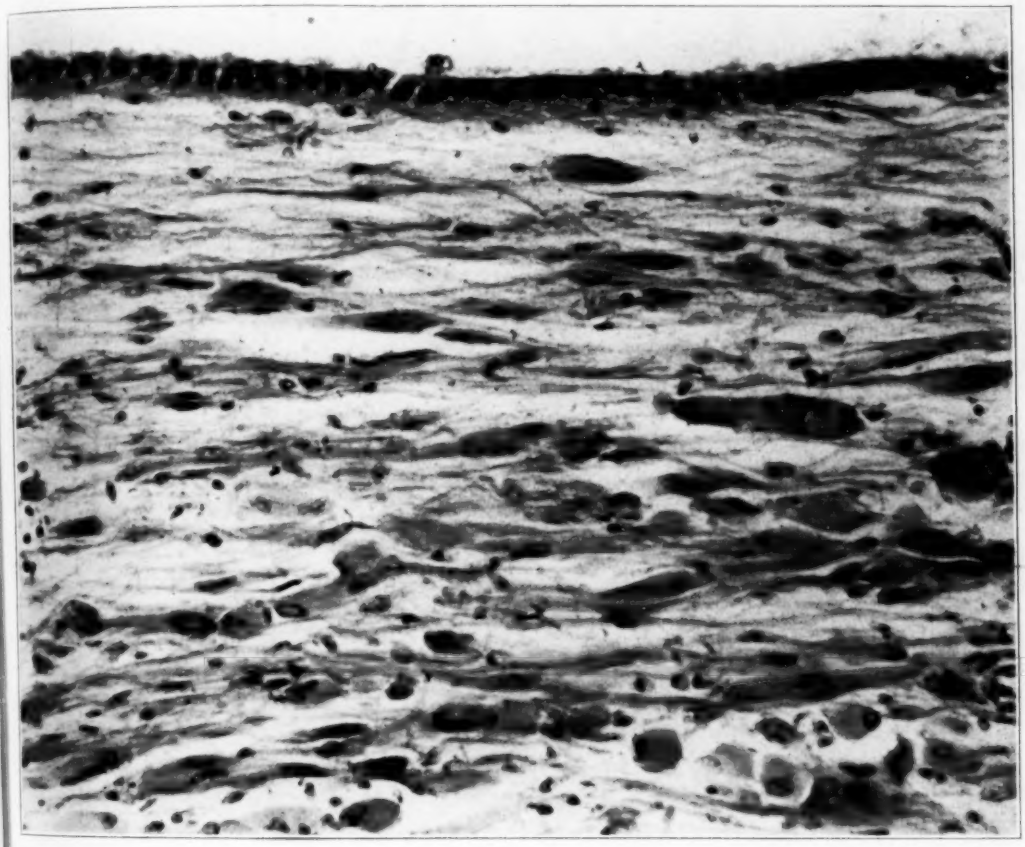

Fig. 17 (case 8).—Section from the periphery of the tumor, illustrating the intact and apparently normal ependyma overlying the tumor tissue. Compare with figure 16. Hematoxylin; × 250.

weakness of both external recti and slight left facial weakness. The right supra-patellar and patellar reflexes were more active than those on the left; the other tendon reflexes and abdominal reflexes were not elicited.

Roentgen examination gave negative results. On lumbar puncture, the fluid was clear, not under pressure, and did not show an increase of cells; the Wassermann reaction was negative.

Course.—In spite of treatment, the coma deepened and on April 20, a right subtemporal decompression was performed under local anesthesia for a suspected left temporal tumor.

The condition grew steadily worse, and the patient died with pulmonary edema one week after the operation.

Autopsy.—The left hemisphere was definitely larger than the right. When the brain was incised, a large grayish purple mass with many hemorrhagic foci was found in the middle portion of the left temporal lobe (fig. 19).

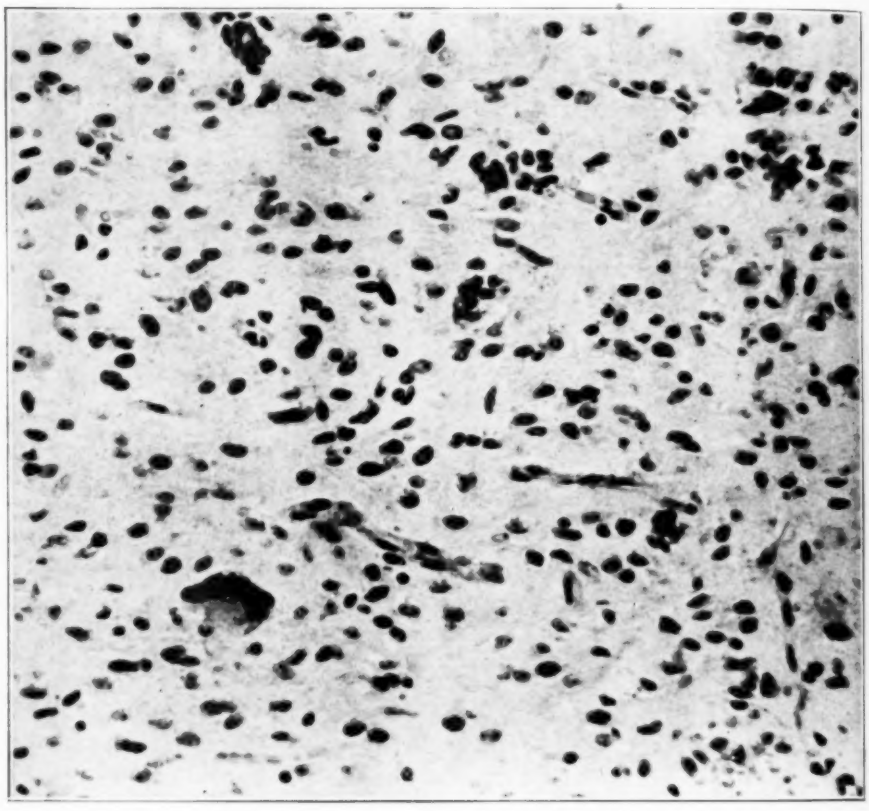

Fig. 18 (case 9).—Numerous undifferentiated glia cells with many multinuclear giant cells of the type usually seen in spongioblastoma. Hematoxylin; × 300.

Microscopic preparations of the tumor presented features typical of glioma (protoplasmatic astrocytoma) (fig. 20).

COMMENT

Incidence.—The ages of the thirty-seven patients in our series were as follows: under 30 years, two; from 30 to 40, five; from 40 to 50, fifteen; from 50 to 60, fourteen and over 60, one.

The patients were therefore usually adults between 40 and 60 years of age, the average age being 45. Twenty-five of the patients were men and twelve were women, so that almost 68 per cent were of the male sex.

Fig. 19 (case 10).—Gross appearance and location of the tumor.

Onset and Course.—When the patients first came under observation, the average duration of symptoms had been forty-nine days; that is, on an average, seven weeks had elapsed between the first symptom and the patient's admission to the hospital. The shortest history was

seven days (two patients) and the longest was four months (three patients), but in most of the cases, the history dated back less than three months.

As the typical histories already given will show, the onset was usually sudden and thereafter the symptoms continued unabated. Headache was usually the first symptom and was frequently accom-

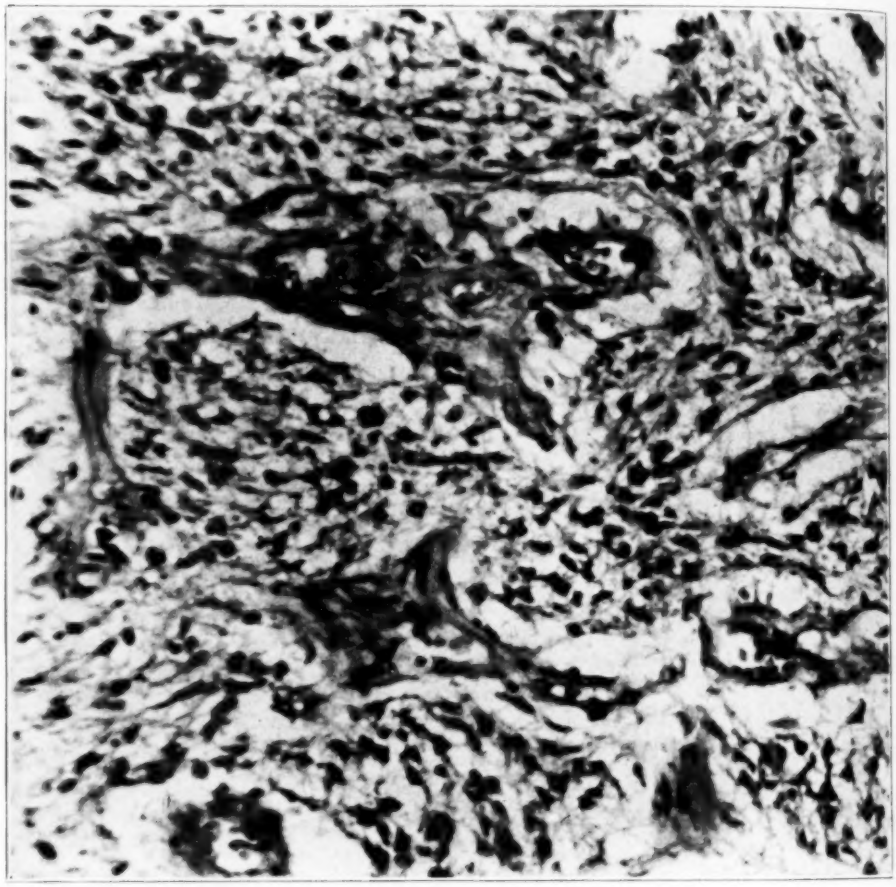

Fig. 20 (case 10).—An average field of the tumor presenting a typical picture of a protoplasmatic astrocytoma (benign glioma). Hematoxylin; × 300.

panied by vomiting. In most of the records, the headache was characterized as of great severity.

In some of the patients the onset was apoplectiform, with sudden dizziness followed by loss of power in one or more extremities. If the symptoms began without headache, severe pain in the head soon appeared and persisted until the patients became stuporous or comatose.

As table 1 shows, headache, vomiting, and drowsiness rapidly progressing to stupor were the early and prominent subjective disturbances.

Most of the patients looked acutely ill, and many of them appeared "toxic," as though severely poisoned by the disease. At first this frequently led to the suspicion that one was dealing with an inflammatory lesion, and the first examination revealed many signs which are often found in encephalitis.

When the patients were first seen (table 1), rigidity of the neck and a Kernig sign, a more or less well marked papilledema, inequality of the pupils and drowsiness or a state of stupor were often present. Not rarely, ptosis, unilateral facial paresis and paralysis of one limb or of the limbs on one side of the body were found.

With these signs, alterations of the tendon reflexes in the affected limbs with a Babinski sign and other evidence of motor involvement

TABLE 1.—*Relative Frequency of Early Neurologic Symptoms and Signs*

	Per Cent
Headache	95
Papilledema	70
Drowsiness, stupor or coma	68
Vomiting	51
Inequality of pupils	41
Vertigo	40
Loss of power in one or more limbs	40
Ptosis	27
Incontinence	27
Rigidity of neck, Kernig sign	24
Strabismus	16
Diplopia	14
Disorientation or delirium	8
Convulsions	8

were almost regularly present, and in the patients who were able to cooperate, sensory disturbances were often noted.

Tenderness on percussion of the skull on the side of the tumor was observed decidedly more often than is usual in tumors of the brain.

The roentgen studies of the skull failed, in all cases, to show any abnormality, and, as was to be expected in an expanding disease of such short clinical duration, there was in no instance any roentgen evidence of increase of intracranial pressure.

Air was injected either directly into the ventricles or by the lumbar route in a number of the cases, and the pneumograms always showed changes in the position and deformities of the outline of some part of the ventricular system, with dilatation of a moderate degree of the contralateral ventricle and alteration of the relative position of the bodies and temporal horns of the ventricle,[8] conditions characteristic of intracranial expanding lesions.

8. Elsberg, C. A., and Silbert, S.: Changes in Size and Relations of Lateral Ventricles in Tumors of the Brain, Arch. Neurol. & Psychiat. **14**:489 (Oct.) 1925.

The study of the blood of the patients showed nothing especially abnormal. As concerns the cerebrospinal fluid, the cell count was within normal limits in about two thirds of the cases, while in more than one third there was a pleocytosis—from 10 to 52 cells per cubic millimeter. The fluid was xanthochromic in about one of every five patients, and in these there was a moderate increase in globulin.

With some exceptions, which will be mentioned later, the progress of the disease was little influenced by medical measures or surgical therapy, and, in the majority of the patients, death occurred within three weeks from the time of admission to the hospital.

Table 2 shows that nine patients, in whom special treatment was not used, died, on the average, 6.3 days after admission; four patients in whom lumbar or ventricular punctures were done for therapeutic purposes died in 19.7 days; two who were subjected to a subtemporal decompression survived, on the average, for 8 days. In twenty-two patients an exploratory cranial operation was performed, sometimes with excision of the tumor en bloc or with removal of the soft new growth by suction, and in all a defect in the bone and dura was left for decompressive purposes. Six of these patients lived for from one to three months, and one recovered and has remained well for more than two years. If the patients in whom death occurred after a longer period are included, the average duration of life from the time of admission into the hospital was 34 days (table 2).

The average length of life from the beginning of symptoms is shown in table 3.

Pathology.[9]—The tumors found at operation or at autopsy were almost always large and were located above the tentorium in thirty-two patients (86 per cent) and in the posterior cranial fossa in five (14 per cent). A number of cases of subtentorial neoplasm have been excluded from this group of tumors, as it seemed probable that the rapid progress of symptoms was due entirely to an acute obstructive hydrocephalus. We are not at all certain that the five patients we have included in our series, in whom the new growth was situated below the tentorium, really belong in this group.

As shown in table 4, the tumor was situated in one temporal lobe and extended into adjacent parts of the hemisphere of the affected side in more than one half of the patients. In about four-fifths (81 per cent), the growth was a spongioblastoma and in less than one sixth, it was a histologically benign gliogenous growth. It is of interest that, with one exception, the gliomas occurred in the patients in whom the new growth was situated in the cerebellum or pons. In four instances,

9. A more extensive pathologic study of the tumors of this group will soon be published by Dr. Globus.

the growths were multiple—a spongioblastoma of each frontal lobe, a glioma of one cerebral peduncle and one in the medulla; a spongioblastoma in the left temporal and parietal lobes and one in the right cerebellar lobe, and a glioma of one frontal lobe and a papillary adenoma of the pituitary gland.

TABLE 2.—*Duration of Life After Admission to the Hospital*

1. Patients in whom only palliative medical treatment was employed		
	2 died in 1 day 2 died in 3 days 2 died in 4 days 1 died in 6 days 1 died in 17 days 1 died in 18 days	average, 6.3 days
2. Patients in whom spinal or ventricular punctures were done		
	2 died in 12 days 1 died in 26 days 1 died in 29 days	average, 19.7 days
3. Patients in whom a palliative decompression was performed		
	1 died in 1 day 1 died in 15 days	average, 8 days
4. Patients in whom a major craniotomy was performed without more than the removal of a specimen of the tumor for verification, or of part of the growth		
	2 died in 11 days 1 died in 13 days 1 died in 17 days 3 died in 20 days 1 died in 22 days 2 died in 25 days 1 died in 29 days 1 died in 39 days 2 died in 54 days 1 died in 56 days 1 died in 75 days 1 died in 82 days 1 died in 92 days	average, 37 days
5. Patients in whom the tumor was removed		
	1 died in 2 days 1 died in 9 days 1 died in 37 days 1 recovered	

TABLE 3.—*Death (in Days) from Time of First Symptom*

19 to 40 days	7 patients
40 to 60 days	7 patients
60 to 80 days	5 patients
80 to 100 days	6 patients
100 to 120 days	5 patients
120 to 140 days	4 patients
140 to 200 days	2 patients
Living and well after more than 2 years	1 patient

Hemorrhage into the tumor occurred in several instances and extensive softening from interference with the vascular supply in two of the cases. "Brain swelling" of the affected hemisphere was frequent.

Differential Diagnosis.—From the description of the onset and the progress of symptoms, the clinical similarity of many of these cases to encephalitis is at once evident; in actual fact, many of the patients were referred to the hospital and were often at first supposed to be

suffering from an inflammatory lesion. After a short period of observation, however, it became clear that the degree of papilledema and the progress of the symptoms were more like those of an intracranial expanding lesion. While papilledema may occur in epidemic and other forms of encephalitis, it is relatively unusual, and it must have been the experience of others, as it has been ours, that most of the patients with papilledema in whom the differentiation between encephalitis and brain tumor is considered, are usually found to be suffering from an intracranial new growth.

In some of the patients of our series, in whom changes in the fundi were not found, the question sometimes arose whether the lesion was entirely a vascular one. The progress of symptoms was unlike that of a vascular lesion; the demonstration of distention of the contralateral

TABLE 4.—*Location of the Thirty-Seven Tumors*

Location of Tumors		
Frontal lobe		5
Temporal lobe		12
Temporal and adjoining lobes		8
Parietal lobe		4
Basal ganglia		2
Pons		1
Cerebral peduncles		1
Cerebellum		4
Multiple Tumors		
Cerebral peduncle, medulla	Spongioblastomas	
One temporal lobe and cerebellum	Gliomas	
One frontal lobe and pituitary	Glioma and papillary adenoma	
Two frontal lobes	Spongioblastomas	
Nature of Tumors		
Spongioblastoma (including transitional and undifferentiated)		30
Glioma		6
Gumma		1

ventricle and other evidences seen in the pneumograms, the later appearance of swelling of the optic disks and the severity of the headache usually pointed to the correct diagnosis.

A few of the patients were so emaciated and looked so ill that metastatic malignant disease was at first considered as a possibility. Usually, the absence of a history of a primary malignant disease in those organs and parts of the body which are often followed by intracranial metastases, the negative result of the thorough search for a primary focus, and the presence of signs of a single lesion made it possible to exclude the diagnosis of a secondary intracranial lesion.

Treatment.—The rapid advance of symptoms of profound involvement of the brain and the extensive lesions found at operation and at autopsy in these patients make it clear that the outlook from any kind of therapy must be poor. Treatment with intravenous hypertonic solutions, palliative lumbar or ventricular puncture and decompressive operations gave equally poor results.

Roentgen treatment did not have any distinct retarding influence on the symptoms. Ewing and others [10] claimed that the more embryonic and "unripe" the type of tumor, the more susceptible it would be to the x-rays. This series, in which the spongioblastomas were so largely represented, does not corroborate this opinion. All of the patients of this group who survived for a sufficiently long period received thorough and intensive roentgen treatment, but in not a single instance was there distinct benefit from this therapy.

Six of the patients in whom an excision of a solid tumor was attempted and a large amount of tumor tissue was removed lived for from one to three months, i.e., the period of survival was longer than after any other method of treatment. Whether better results can be obtained by the excision of the growth with the electric cautery knife will have to be determined in the future. We have, as yet, had an insufficient experience with this method of removal of infiltrating tumors of the brain.

In two patients, the tumor was of jelly-like consistency and was removed by suction. One of the patients died and the other was living and well more than two years after the operation. As the histories of these two patients are of considerable interest, they are given in abstract.

CASE 11.—*Two months' history of cerebral symptoms. Craniotomy and removal by suction of soft tumor in left temporal lobe. Roentgen therapy. Complete recovery. Well twenty-six months later.*

History.—N. A., a man, aged 33, was admitted to the hospital, May 4, and discharged, June 8, 1926. Four years before admission to the hospital, he was struck over the back of the head by a blackjack. He had been treated for sinus trouble ten months before admission. Two months before admission, on account of severe headache, the tonsils were removed. Soon afterward, the headache became severe and continuous. For several weeks he had had attacks of dizziness and nausea and had had difficulty in naming objects. For the past week he had been drowsy and disoriented.

Examination.—The patient was somewhat euphoric and seemed dazed. The pupils were unequal but both were somewhat dilated. There were double choked disks of 3 diopters; slight facial weakness, supranuclear in type; distinct weakness of the right upper and lower limbs with diminution of the tendon reflexes but with ankle clonus and a Babinski sign. Sensation, as far as could be tested, appeared normal. The patient had a mixed aphasia. There was distinct rigidity of the neck. Tenderness was present on percussion of the skull over the temporal region on the left side.

The Wassermann reaction of the blood and spinal fluid was negative. Roentgen examination gave negative results.

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10. Ewing, J.: Tumors of Nerve Tissue in Relation to Treatment by Radiation, Am. J. Roentgenol. **8**:497, 1921. Bailey, P.; Sosman, M. C., and Van Dessel, A.: Roentgen Therapy of Glioma of the Brain, Am. J. Roentgenol. **19**: 203, 1928.

Operation.—On May 11, 1926, an osteoplastic flap was turned down on the left side (by one of us, C. A. E.). The dura was moderately tense. On incision of the dura, a brownish discoloration was exposed in the upper part of the temporal lobe. Incision of the brain over this area revealed a tumor of jelly-like consistency extending deeply into the temporal lobe. A large quantity of tumor tissue was removed by suction, and the walls of the cavity were cauterized with Zenker's solution. The dura was partially closed. The base of the bone flap was rongeured away for decompressive purposes. The bone flap was replaced and the galea and skin were sutured.

Course.—Recovery from the operation was uneventful. The disturbances in speech cleared up rapidly; the reflexes were equal on the two sides, and the disks were flat when the patient was discharged from the hospital four weeks after the operation. For a number of months, he received deep roentgen therapy. Except for "fits of temper," he was well and free from symptoms more than two years after the operation.

Histology.—The histologic picture of the tumor was that of a spongioblastoma multiforme, characterized by the presence of many spongioblasts aggregated with fairly extensive islands surrounded by wide vascular channels (fig. 21). Glia cells of a more differentiated type alternated with the prominent giant-like spongioblasts (fig. 22).

NOTE: In September, 1928, the patient returned with a recurrence of his symptoms—papilledema, aphasia, hemiparesis; after the removal of much tumor tissue by suction, the neurologic disturbances again disappeared. Examination of the tissue removed at operation again showed it to be a spongioblastic growth. The cellular make-up, however, was more of the type seen in figures 24 and 25.

The satisfactory result in this patient was considered to have been due not only to the operation but to the thorough roentgen therapy; the patient was presented at a meeting of the New York Neurological Society, as being greatly benefited by roentgen treatment. This point of view was probably erroneous because of the following experience we had about one year later:

CASE 12.—*Spongioblastoma of the right parietal lobe. Craniotomy and removal of soft tumor by suction. Death within twenty-four hours. Autopsy demonstrated that the tumor had been completely removed.*

History.—Salvatore A., a man, aged 31, was admitted to the hospital on March 3, and died March 5, 1927. Seventeen days before, having been in good health up to that time, he awoke with severe frontal headache and cramplike pain in the left upper and lower limbs. He vomited soon afterward. The headache and pain continued, but he was out of bed off and on for one week. For ten days before admission he was bedridden, and the headache was severe. Two days before admission he became drowsy, and for the last twenty-four hours he was incontinent of urine.

Examination.—On admission the patient was stuporous, so that he could be aroused only with difficulty. Both fundi showed early papilledema. The right pupil was larger than the left, and both pupils reacted sluggishly to light and in accommodation. There was distinct paresis of the facial musculature on the left. The limbs on the left side were paretic, with bilateral ankle clonus and Babinski confirmatory signs, more marked on the left. The abdominal and cremasteric reflexes could not be elicited.

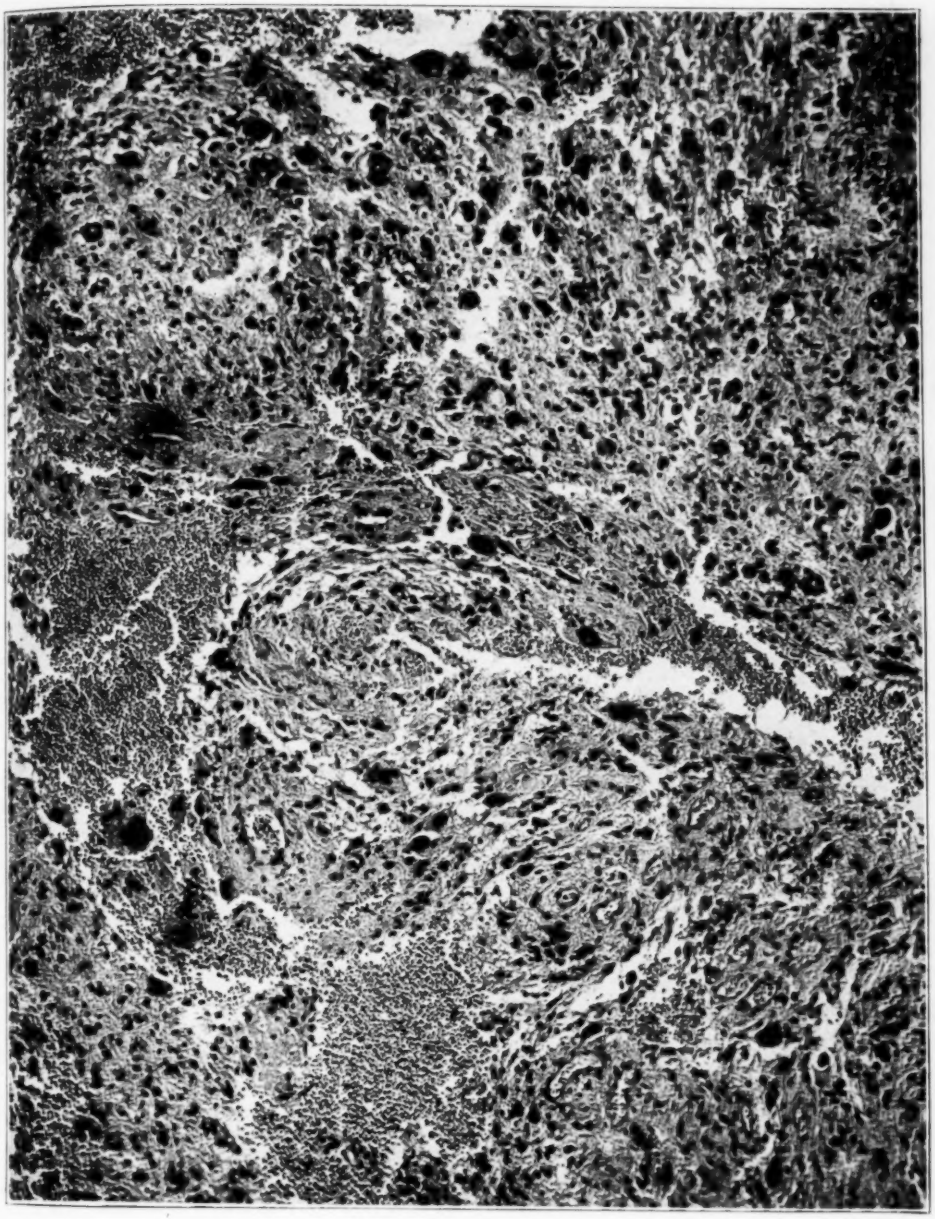

Fig. 21 (case 11).—General appearance of an average microscopic field. The large number of giant cells should be noted. Hematoxylin; × 300.

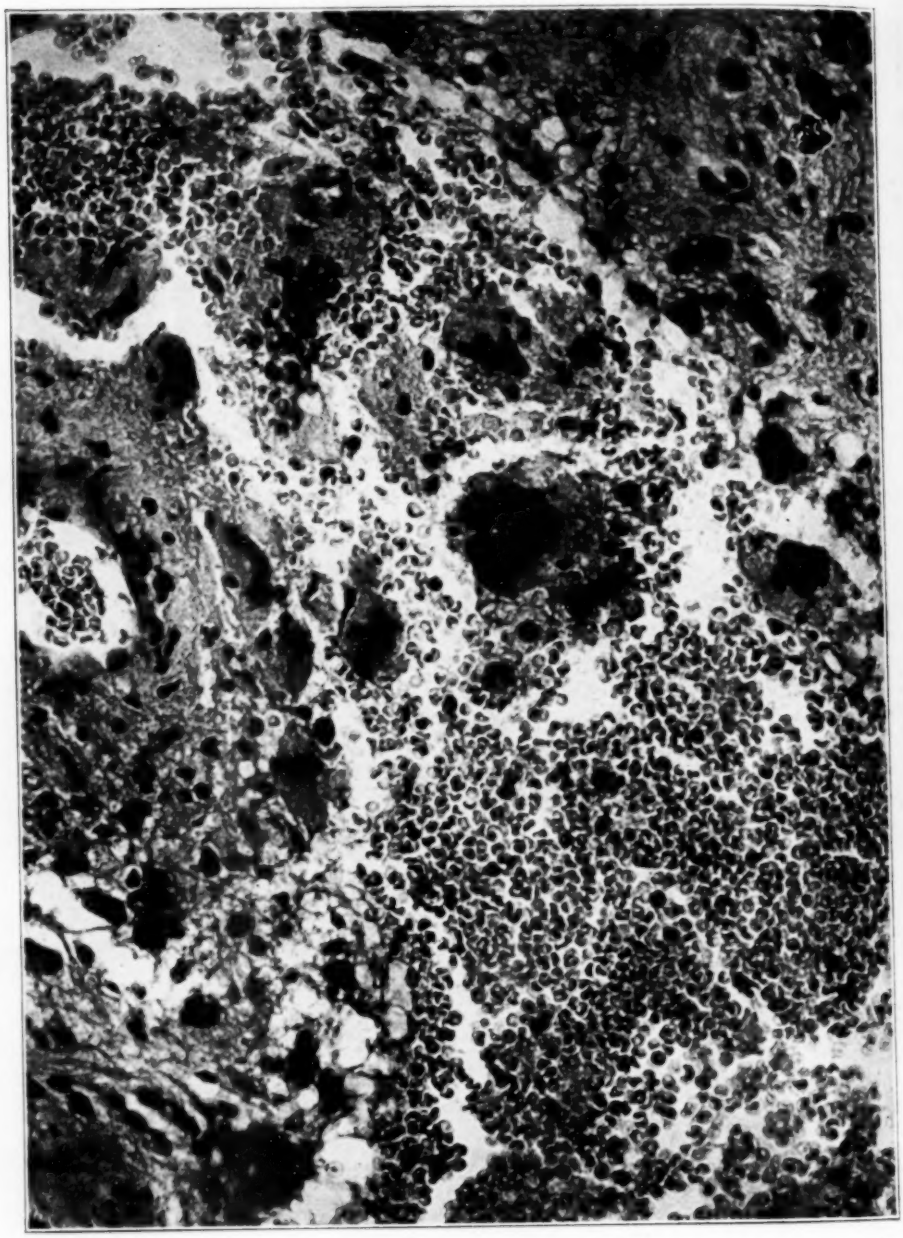

Fig. 22 (case 11).—Higher magnification of an area in field (fig. 21), showing the character of the multinuclear giant spongioblasts. Hematoxylin; × 800.

Course.—On the following day left homonymous hemianopia and astereognosis in the left hand seemed to be present. The spinal fluid was under increased pressure (200 mm.), but was normal in all respects. Roentgen examination gave negative results.

The patient became more stuporous and was operated on as an emergency. Under local anesthesia, a large flap was turned down by one of us (C. A. E.),

Fig. 23 (case 12).—Gross appearance and location of the cavity left after the removal of the tumor.

exposing the right frontal, parietal and temporal lobes. On incision of the dura, a large brownish colored tumor lying in the parietal lobe was exposed. Its margins were fairly well limited. The growth was incised and found to be of a soft consistency and not vascular, so that it could be removed by suction. More and more of the growth was removed by suction until a cavity 6 cm. in length and 4 cm. in depth remained. Its walls were cauterized with Zenker's solution. There

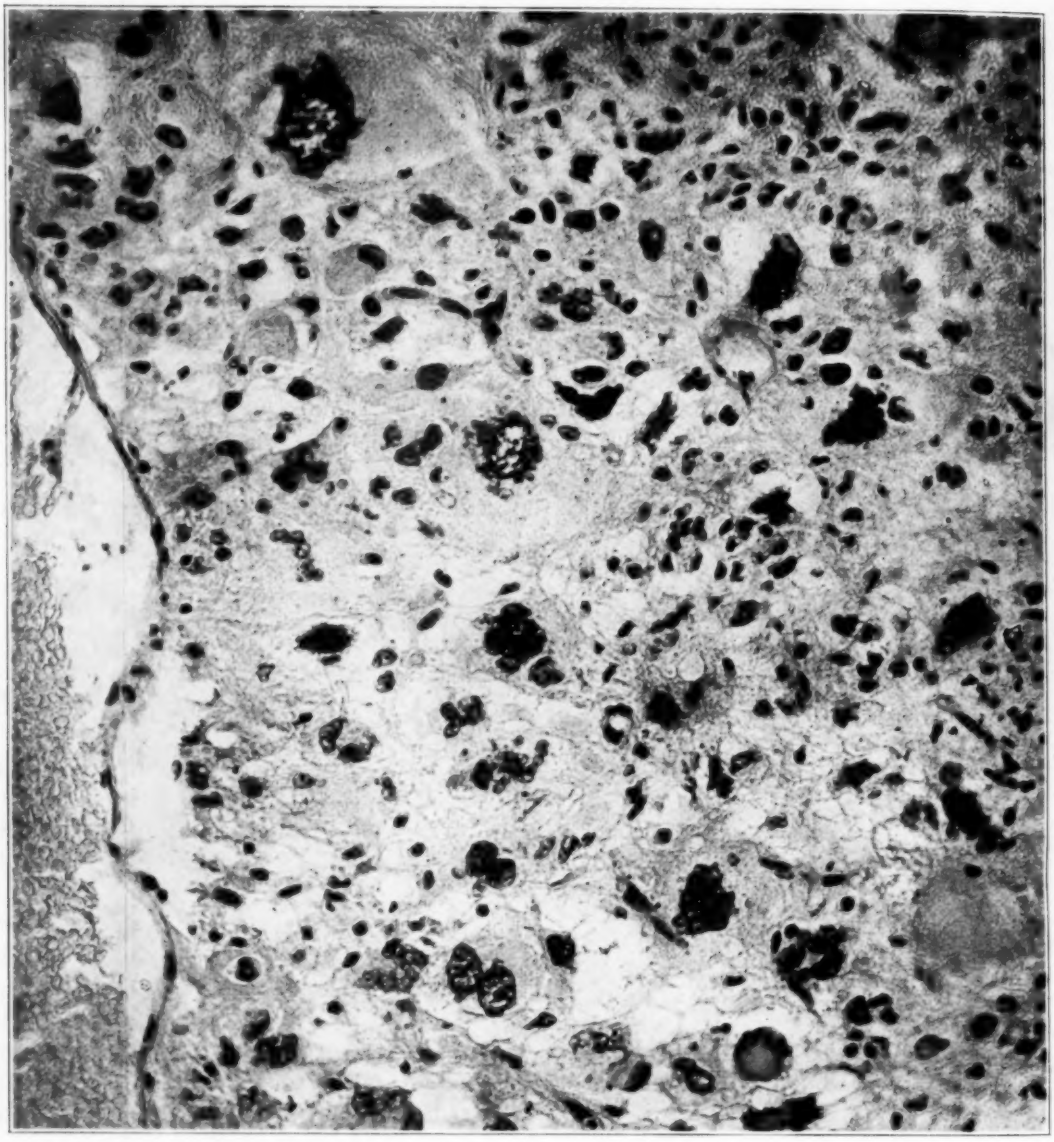

Fig. 24 (case 12).—General appearance and arrangement of cells. The exceedingly large number of multinuclear giant spongioblasts should be noted. Hematoxylin; × 600.

was little bleeding during all of the manipulations, but the patient stood the operation poorly. The dural incision was closed, the bone flap returned into place and the soft issues sutured.

In spite of intravenous therapy and energetic stimulation, the patient's condition did not improve. Death occurred five hours later.

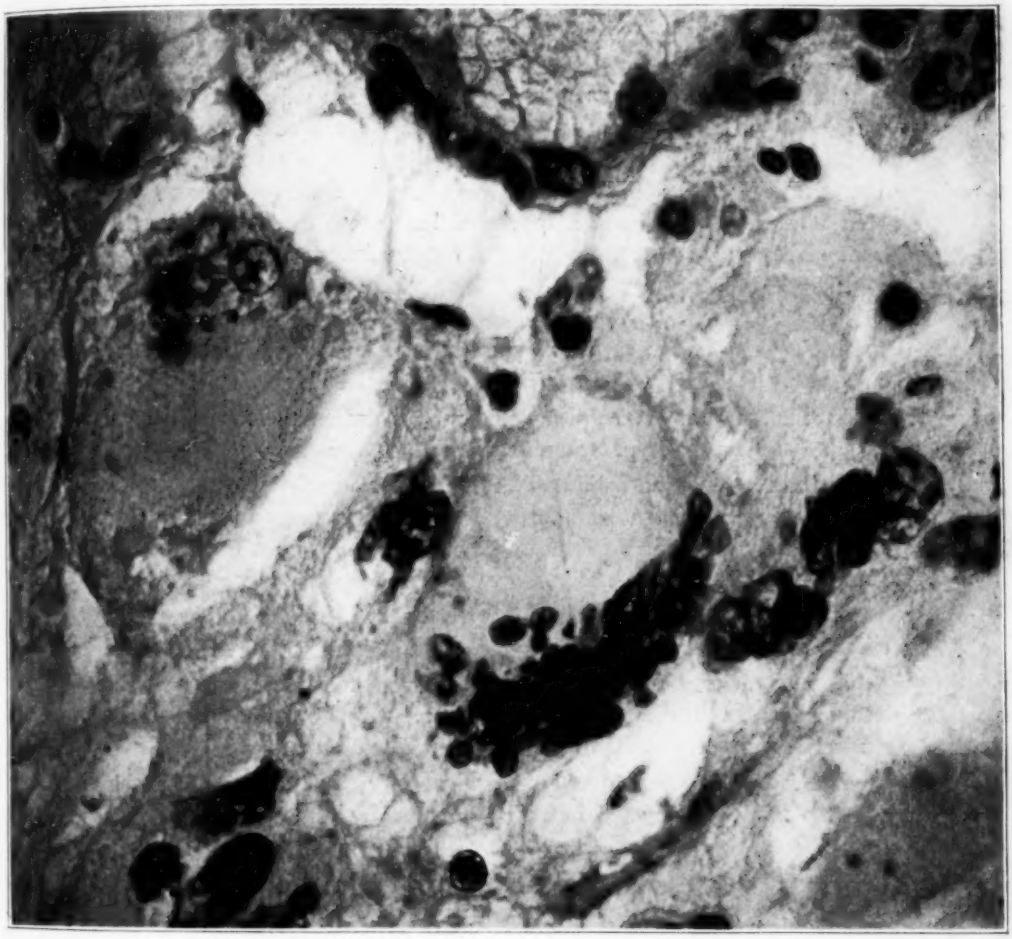

Fig. 25 (case 12).—A small area under higher magnification, illustrating the nuclear aggregations in the giant spongioblasts; × 1,000.

Autopsy.—The right hemisphere showed a crater-like cavity almost completely replacing the parietal lobe. The wall of the cavity was smooth, apparently free of tumor tissue and only here and there contained a small amount of necrotic tissue. The brain tissue surrounding this cavity showed marked edema. The latter was most pronounced in the region of the precentral gyrus, which was undermined by the cavity (fig. 23).

The histologic picture of the tumor was striking. The dominant type of cell was the multinuclear giant cell (fig. 24). The unusually large size of the cells and the large number of nuclei (fig. 25) were uncommon even in spongioblastoma. The nuclear aggregations were such as to suggest rapid proliferation. This may be regarded as the explanation for the rapidity of the clinical course. Of interest is the fact that special stains (such as the Hortega-Globus method) brought out many microglia cells.

The complete removal of the growth by suction was a surprise to us, for the operator had supposed that some tumor tissue had been left. This case led us to revise our views of the case in which the patient has remained well for more than two years. As the consistency and the histologic character of the tumor were the same in both instances and the removal by suction followed by cauterization with Zenker's solution was similar, the good result may well have been due to the thoroughness of removal rather than to the roentgen therapy.

SUMMARY AND CONCLUSIONS

1. A group of cases of tumor of the brain has been described in which the onset was acute and the clinical course was rapid.

2. The patients were most often men, between the ages of 40 and 60, and were usually profoundly affected by the disease.

3. The prominent symptoms and signs were headache, vomiting, papilledema, irregularity of the pupils, marked changes in motor power and reflexes, tenderness of the skull on percussion over the site of the growth, drowsiness or stupor, rigidity of the neck and a Kernig sign.

4. The tumors were usually large spongioblastomas and were most often located in or near a temporal lobe.

5. The differential diagnosis from acute encephalitis or a vascular lesion was often difficult at first. Some of the cases presented a clinical picture like that of metastatic malignant disease.

6. Treatment, surgical and otherwise, gave equally poor results, but life was most prolonged by partial or complete removal of the tumor.

OSTEOMA OF THE SPINAL ARACHNOID WITH PSAMMOMATOUS DEGENERATION *

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Whereas most authors describe deposits of calcium in the cerebrospinal meninges, including psammomas and osteophytes, as of not uncommon occurrence, all are agreed that osteomas and chondromas are rare. Burle and Morel,[1] in reporting an osteoma of the dura, quoted Devay as being able to find reports of only ten cases in the literature in 1908, when the latter reported a case at the Société Nationale de Médecine.

In Lewandowsky's Handbuch,[2] Flatau made a careful classification of 213 cases of tumor of the spinal cord, using Schlesinger's statistics, but did not mention osteomas. Ernst, in Aschoff's Treatise,[3] stated that chondromas are rare but did not mention osteoma. Oppenheim,[4] in his large series, did not mention the osteoma, though he spoke of the psammoma as a comparatively rare tumor of the spinal cord. Ziegler [5] described psammomas as a calcareous degeneration occurring in sarcoma of the pia.

In the classic Traité of Cornil and Ranvier,[6] who studied the psammoma critically and called it sarcoma angiolithique, there is no mention of osteoma of the meninges.

Bouchard,[7] in his extensive article on the spinal cord (1874), which takes up practically the whole of volume 8 of the large Dechambre

* Submitted for publication, July 7, 1928.

* From the service of Associate Professor G. Roussy and Dr. J. Lhermitte, of Hospice Paul Brousse, Villejuif, Seine.

1. Burle and Morel: Ostéome de la dure-mère, Lyon méd. **114**:1095, 1910.

2. Lewandowsky, M.: Handb. d. Neurol. **2**:642 and 668, 1914.

3. Ernst, P., quoted by Aschoff, L.: Pathologische Anatomie, Jena, 1909, vol. 1, p. 731.

4. Oppenheim, H.: Text Book of Nervous Diseases, ed. 5, Chicago, Chicago Medical Book Company, 1911.

5. Ziegler, Ernst: Lehrbuch der allgemeinen und speciellen pathologischen Anatomie für Aerzte und Studierende, Jena, G. Fischer, 1895.

6. Cornil and Ranvier: (Grand Traité), 1907, vol. 3, p. 85 (Nageotte and Riche).

7. Bouchard: Moelle, Dict. d. Soc. Méd., 1874, second series, vol. 8, p. 665.

Dictionary, did not mention osteoma. Psammoma as "sarcome angiolithique" is given the usual prominence. Oustaniol,[8] in his commendable thesis on tumors of the spinal meninges (1892), stated that the psammoma is in fact the special tumor of the meninges. He, also, did not mention osteoma.

Ball and Krishaber[9] studied the question from a clinical point of view in their article on the brain. They reported two cases, but they were osteomas of the brain proper and did not relate to the meninges.

Archambault,[10] in his article on the meninges, stated that cartilaginous tissue and ossification in the form of small tumors or plaques of bone are sometimes found in the meninges.

Roussy and Leroux[11] placed the psammomas among the disputable endotheliomas and classified them "more properly" as fibromas or fibrosarcomas arising from the connective tissue of the meninges. They described them as tumors consisting of connective tissue (fibroblasts) with vessels of an embryonic or adult type, with the formation of the characteristic concentrically arranged hyaline or calcareous cells. Furthermore, they believed that these whorled cells originate by a proliferation of the endothelium of the vessel walls; these undergo hyaline degeneration, and later calcium is deposited forming the so-called psammoma bodies.

Among the American authors, Delafield and Prudden[12] described osteophytes, plates and globular masses of bone found in the meninges, and attributed them to local inflammatory processes. They spoke of the psammoma as a calcium deposit in fibrous, sarcomatous or endotheliomatous tissues. Osteoma and chondroma are rare according to these authors.

Adami and McCrae[13] stated that osteomas, as typical bony tumors growing independently of the tissue in which they exist, are rare. They designated as "metaplastic ossification" the osteophytes and plaques of bone found in the meninges and attributed them to inflammatory processes. "So-called osteomas" these authors continued, "are, strictly speaking, osteophytes showing no inherent tendency to grow. They are

8. Oustaniol, Jules-Gaspard: Contribution à l'étude des tumeurs des méninges rachidiennes; anatomie pathologique; symptomatologie; traitement chirurgical, Oustaniol, Thesis Univ. Paris, Steinheil, Paris, 1892.

9. Ball and Krishaber: Cerveau, Dechambre Dict. **14**:437, 1873.

10. Archambault: Méninges, Dict. encycl. d. sc. méd. **6**:574, 1873.

11. Roussy and Leroux: Diagnostique des tumeurs, Paris, Masson & Cie., 1921.

12. Delafield and Prudden: Text Book of Pathology, ed. 8, New York, William Wood & Company, 1908, p. 867.

13. Adami and McCrae: A Text Book of Pathology for Students of Medicine, Philadelphia, Lea & Febiger, 1914, p. 595 and 596.

plaques of osteoid tissue of metaplastic origin from the connective tissue." These authorities also regarded psammomas as endothelial tumors.

MacCallum [14] stated that osteomas, or bony tumors in which the bone grows independently without a limited aim, are rare. He did not mention osteoma of the meninges.

Ewing [15] described the psammoma as either a cellular and vascular form of sarcoma or a less cellular sclerosed tumor with much calcification. He mentioned osteomas as appearing as nodules of bone in the cerebral and spinal arachnoid.

Cushing and Weed [16] described a case of dural endothelioma with psammoma formation and again ossification in the arachnoid of a patient with a temporal endothelioma. The authors concluded that these tumors arise from the mesothelium of the arachnoid and not from the dura.

O'Kelly [17] reported a psammomatous degeneration in an endothelioma or perithelioma. It had not caused symptoms and was found at autopsy.

More recently, McKendree and Imboden [18] recorded a case of ossification of the meninges in which the condition was diagnosed from clinical and roentgenographic examinations. The patient was a woman, aged 49, who had suffered for years from cerebral symptoms. These conditions appear to be more common in the female sex.

In 1928, Lambert Rogers [19] reported a spinal meningioma containing bone, in the case of a school girl, aged 16. From the histologic description of this tumor, and his illustration, it appears to be similar to the one here described.

Thus, after a search of the available literature I have found but one case report of a tumor of the meninges showing the same picture as in the case here reported.

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14. MacCallum, W. G.: A Text Book of Pathology, Philadelphia, W. B. Saunders Company, 1920.

15. Ewing, James: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, 1919, p. 424.

16. Cushing, Harvey, and Weed, Lewis H.: Studies of the Cerebro-Spinal Fluid and its Pathway: IX. Calcareous and Osseous Deposits on the Arachnoidea, Bull. Johns Hopkins Hosp. **26**:367 (Nov.) 1915.

17. O'Kelly, W. D.: Notes on a Psammoma of the Dura Mater, Dublin J. M. Sc. **4**:26 (Jan.) 1921.

18. McKendree, Charles A., and Imboden, Harry M.: Ossification of the Meninges, Arch. Neurol. & Psychiat. **6**:529 (Nov.) 1921.

19. Rogers, Lambert: Spinal Meningioma Containing Bone, Brit. J. Surg. **15**:675, 1928.

REPORT OF CASE [20]

History.—A housewife, aged 74, was admitted on Feb. 7, 1918, to an old peoples' home, on account of simple decrepitude. Menstruation had begun at the age of 11, and the menopause had occurred at the age of 48. She was married, but did not have any children. There had been one miscarriage. Her husband was in good health.

After the patient was admitted to the home, she presented symptoms of intermittent claudication. One evening, while washing dishes, she was suddenly seized with weakness of the legs and fell on her knees. This paraplegic state was temporary, lasting only five minutes, after which she was again able to stand and continue her work. Since this episode, the patient had constantly suffered from slight difficulties in gait, especially on the right side; "My feet feel like a rag."

During the evening of Nov. 4, 1919, she was again suddenly seized with difficulty of locomotion. She was unable to walk, though she could stand; she noticed that her right hand felt heavy and was awkward. She could no longer feel objects placed in her hand. She had to be carried upstairs to her room and later it became necessary to transfer her to the infirmary ward.

Examination.—On November 7, there was a distinct improvement in the monoplegia of the right arm, but great difficulty was still manifested in the finer movements of the hand and fingers. (There was a marked retardation at the beginning of a given movement, and toward the end the execution was rapidly accomplished.) There was a considerable diminution in muscular strength, the dynamometer registering 10 degrees on the left, and 2 on the right side. An incomplete paraplegia was present in the lower extremities. The patient was able to stand but needed assistance. She was able to walk with nurses supporting her on each side. There was a partial flexion of the right lower extremity, which was weaker and spastic.

The knee and ankle reflexes were active, especially on the right. The biceps, pronator and triceps reflexes were also active. In the plantar reflexes, there was a frank extension on the right; on the left there was also a Babinski sign, but it was less distinct.

Hypesthesia to touch and pain was present, but was less marked on the right side. There was not any disturbance in deep sensation, stereognostic sense or sense of position. Sphincter disturbance was not present.

There was a slight anisocoria, the left pupil being one quarter the size of the right. The reactions to light and in accommodation were normal.

There was no disturbance of the auditory, olfactory or gustatory functions.

The heart was normal.

There was a large fibromyoma of the uterus; an endometritis with a "café au lait" colored exudate was present.

The urine contained neither sugar nor albumin.

On Nov. 11, 1919, there was a suggestion of a Babinski reaction on the right side; the Hirschberg-P. Marie reaction was clearcut. On the left, there was no reaction.

The systolic blood pressure was 130; the diastolic, 70.

Course.—The patient was discharged from the hospital ward and returned to her pavilion.

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20. I wish to express my gratitude to Dr. Lucien Cornil, Assistant Neurologist to Hospice Paul Brousse, and Préparateur in Pathologic Anatomy at the Faculty of Médecine of Paris, for his kindness in permitting the use of his excellent clinical notes.

On May 20, 1920, she was readmitted to the sick ward. For the past two months there had been progressive paraplegia, which since two days before the time of writing, had become practically complete. Walking and standing had become impossible. In both lower extremities there were contractures in extension. Voluntary movement of the feet and toes was impossible. She could slightly flex the leg on the thigh on the right side. There was a fixed adduction of the thighs when at rest. The reflexes were lively in the lower extremities, especially on the left. There was a suggestion of an ankle clonus. A double Babinski sign was present, very clearcut on the left on slight irritation. The medullo-automatic reflex (sign of P. Marie-Foix) was well developed.

The uterine discharges had become bloody and abundant for the past month.

On June 10, there was a tactile anesthesia for an extent of two fingerbreadths above the limit of the previous day.

On June 14, the muscular force in the upper extremities was equal on the two sides. Monoplegia was no longer demonstrable. Movements of flexion and extension were attempted on the right, but were possible even to a less extent on the left.

During the night, there were rhythmic movements of flexion in the lower extremities, with cramping pains. There was a tendency to clonus on both sides. There was a hypertonicity of both lower extremities, more marked on the right. The tendon reflexes were slightly more lively on the right. The automatic reflex was frank. On plantar stimulation, there was a prompt drawing up of the lower extremity. Stroking over the internal aspect of the right thigh caused extension of the right leg, with bilateral adduction. The same stimulation on the left caused flexion of the leg on the thigh and of the foot on the leg with a slight internal rotation of the foot. The limit of the reflex of defense was at the twelfth dorsal segment, while the upper limit of anesthesia varied with each day, at the sixth, seventh or eighth dorsal segments.

There was tactile, pain and temperature anesthesia over the dorsum of both feet and over the external aspect of both legs. There was a loss of sense of position for the great toe, but not for the foot and leg.

On June 21, there was a tendency to clonus of the right ankle.

On June 22, the patient became suddenly comatose. There was an absence of spontaneous movements. The Babinski sign was present on both sides. The reflex of defense was bilateral. The tendon reflexes were lively. The ankle clonus was more distinct on the right. The patient complained of pain in the legs.

On June 26, there was a tendency to ankle clonus on the right on percussion of the tendo achillis. The tendon reflexes on the left were more feeble, especially the ankle reflex. The defense reactions were still present.

On June 27, an ankle clonus was present on the right. There was an increased reflex activity of both lower extremities. Pain was not present. The patient died on July 1.

Laboratory Reports.[21]—On June 15, Ambard's constant was 0.09. The urea of the blood was 0.33 Gm. per liter. On June 15, the pressure (Claude's manometer) of the spinal fluid was 21 cm. of water at the beginning, and 5 cm. after the tap. Eight cubic centimeters of clear fluid was removed; it presented: 20 leukocytes in each field; albumin 5 Gm. (according to Sicard's method, about 2 Gm.); an alkaline reaction; a negative Bordet-Wassermann reaction. Examination of the blood showed: erythrocytes, 5,200,000; leukocytes, 20,000. The differential count showed:

21. By courtesy of Dr. Edouard Peyre, Biochemist to Hospice Paul Brousse, Villejuif, Seine.

polymorphonuclears, 70 per cent; mononuclears, 10 per cent; lymphocytes, 11 per cent; transitionals, 8 per cent; abnormal cells, 1 per cent, and hemoglobin, 60 per cent.

Autopsy (dorsal view).—The tumor was a small, hard, slightly nodular mass, somewhat resembling a navy bean in shape and size, measuring 17 by 10 by 10 mm. Its long axis was parallel with the long axis of the cord. The free surfaces of the tumor were smooth. It was attached to the dura mater from which, however, it could be peeled off without tearing. It was not attached to the cord. This hard mass pressed against the cord at and above the level of exit of the ninth dorsal spinal nerves and more directly on the posterior root of the left member.

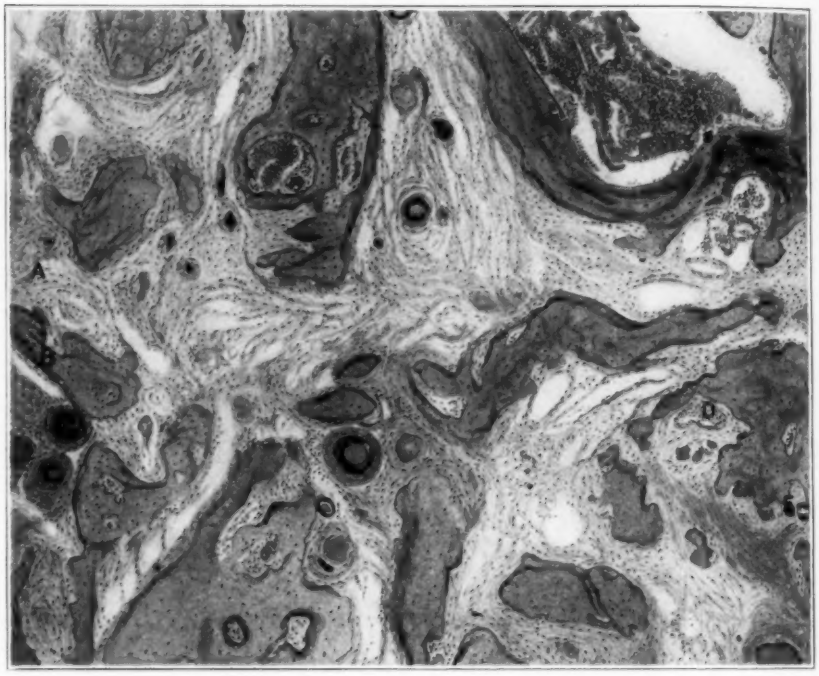

Fig. 1.—Connective tissue stroma with formation of osteoid tissue and numerous psammoma bodies; *A*, connective tissue undergoing change; *B*, osteoid tissue; *C*, psammoma bodies; *D* and *E*, newly formed blood vessels. Mallory stain modified by Masson and Leroux; × 62.

The cord was compressed as a consequence and was slightly softer in consistency at this level. Section here showed the cord thinned and misshapen. It was pushed to the right by the tumor mass and curved around it, making a counter swerve to the left below the tumor before it again assumed a central position.

On cutting, the tumor was found to be calcified and crumbled under the knife, with the liberation of several small, irregular calculi of stony hardness. There was a thin, bony crust surrounding the tumor, very hard and of variable thickness, up to 1.5 mm.

The divided parts were decalcified in acid and then embedded in celloidin and sectioned. Sections of the cord were likewise made at the level of the tumor and

above and below. These were prepared by the methods of Bielschowsky, Weigert (Loyez), hematin and eosin, and Mallory (modified by Masson and Leroux). Sections of the tumor were stained with hematin and eosin and the aforementioned modified Mallory stain.

The tumor consisted of a connective tissue stroma distinctly separate from an osteoid parenchyma. The connective tissue, richly supplied with numerous vascular channels, was in an active state of proliferation and metamorphosis. Where it bordered on the bone tissue, the cells were generally slightly elongated and more closely packed and took a more pronounced basic stain. The vessels, which were found only in connective tissue, were supplied with an intima that was readily

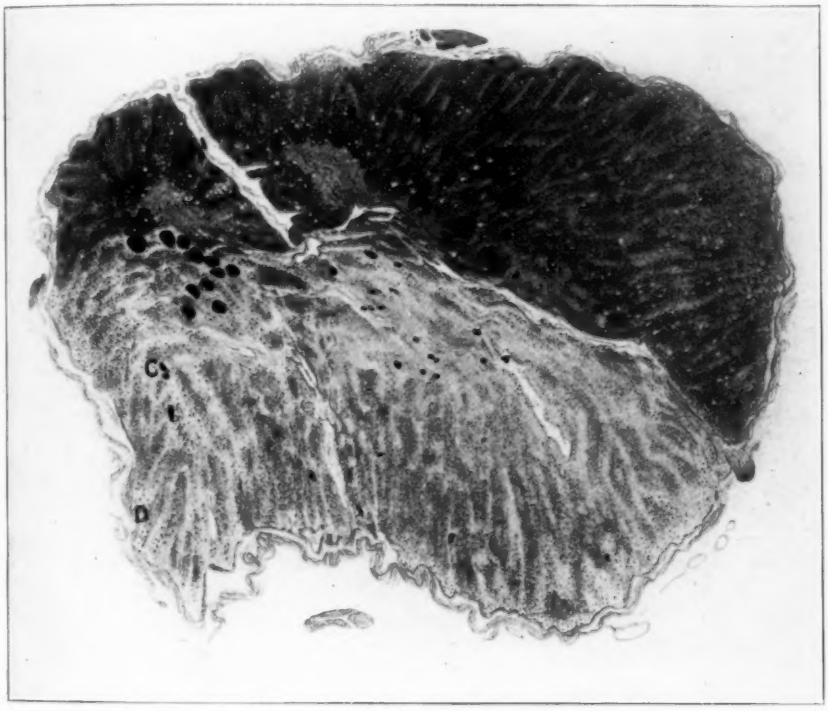

Fig. 2.—Compression myelitis at level of tumor—ninth dorsal pair—showing advanced degeneration, increase of connective tissue and marked distention of the vessels; *A*, degeneration of anterior horn cells; *B*, distended vessels; *C*, degenerated area; *D*, thickened pia. Loyez method for myelin sheath; × 19.

distinguishable in the smallest branches. Throughout the tumor, the thickened, degenerated and calcified vessels could be seen, which constituted the psammoma bodies. Sometimes the walls of the vessels were merely thickened; in other instances hyaline degeneration was already present, with the lumen greatly restricted or already obliterated, and finally the calcified, fully formed psammoma body could be seen.

The bony part of the tumor consisted of typically constituted bone cells with encapsulated nuclei. It was arranged in irregular masses and projections growing in all directions, constituting, apparently, an independently growing osteoma (fig. 1).

The cord was compressed and distorted at the level of the tumor, especially on the left side (fig. 2). It was smaller, and the meninges were proportionately wrinkled and reduplicated. The meninges, moreover, were considerably thickened; in places they were almost twice their normal thickness, revealing compression meningitis.

In the cord proper, the most striking changes were the marked edema and the secondary degenerations. The cord was slightly reduced as a whole; the right half was relatively enlarged, being approximately one and a half times as large as the left, which was directly pressed on by the tumor. Numerous blood vessels, enormously dilated, stood out boldly on the left; there was also some dilatation of the vessels on the right, though this was not nearly so marked.

The cells of the anterior horns were destroyed to a great extent, though a few were still present; they were more numerous on the right.

The Weigert preparations (method of Loyez) showed extensive degeneration of the myelin sheaths, which were replaced by connective tissue that had proliferated freely.

The degeneration of the tracts did not extend beyond the level of the tumor, either upward or downward.

SUMMARY

A case of osteoma of the spinal arachnoid was found at autopsy in a woman, aged 74 years. There had been symptoms of claudication and later of pressure with a syndrome of physiologic section of the cord, similar to those described by Lhermitte,[22] as myelitis extending over a period of more than eight months.

The tumor consisted of a rounded mass, 17 by 10 by 10 mm., the shape of a bean, which on histologic examination was found to be an osteoma in which psammomatous degeneration of the vessels could be demonstrated.

22. Lhermitte, J.: La section totale de la moelle dorsale, Bourges, 1919, p. 229.

DISSEMINATED SARCOMATOSIS (MELANOBLASTOMA?) OF THE CENTRAL NERVOUS SYSTEM AND THE MENINGES

REPORT OF A CASE *

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Cases of malignant tumors of the spinal meninges without invasion of the spinal cord have been described repeatedly. An additional case is here recorded in which the subarachnoid space was densely infiltrated with tumor cells, especially around the cauda equina, while the spinal cord exhibited degenerative changes not unlike those seen in tabes dorsalis. An attempt has been made to follow up the genesis of such changes in the cord and to show what bearing, if any, they may have on the pathogenesis of this disease.

REPORT OF CASE

History.—A man, aged 37, seen by one of us (P. B.) in consultation with Dr. B. Beers at the Grant Hospital, Sept. 13, 1927, complained for two months of pains in the legs and shoulders. About the middle of August, he became unable to walk without help. At about the end of this month, he complained of diplopia, generally vertical, and urinary disturbances—first, incontinence and for the past four days, retention. He had vomited frequently for six weeks.

Examination.—The right pupil was sluggish to light; the left was normal. The vision was poor in the right eye, so that the patient could read only headlines with this eye. Diplopia was present when he looked to the left or downward. The right disk was blurred and much swollen; the left was slightly blurred, but scarcely swollen. The neck was somewhat stiff. Gross cranial nerve palsy was not present. The left leg was weak, and considerable ataxia was present in the arms and legs, more so in the right leg. The superficial reflexes were normal, while the tendon reflexes were absent throughout. Sensibility was normal.

Course.—The legs grew weaker and more painful. Several attempts at a lumbar puncture were unsuccessful; fluid was not obtained, though the needle appeared to have entered the canal. The headache ceased within a week, but the paralysis in the legs was complete by the end of September. On September 30, Dr. Kraft found a swelling of 2 diopters in the right disk, and the field of the right eye, espe-

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* From the laboratories of the Research and Educational Hospital and the Illinois State Psychopathic Institute.

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cially the temporal part, was much contracted. After October 1, there was gradual loss of sensation in the lower extremities and buttocks, and bed sores began to develop. The right eye became blind and the right pupil ceased to react to direct light, but reacted consensually. Vision in the left eye failed gradually during November. Pain in the legs, particularly in the left foot, remained troublesome even after sensibility was almost lost in both lower extremities. The patient gradually failed and died, Dec. 27, 1927.

Summary of the Clinical Examination.—Progressive weakness and pain in the lower extremities which resulted in paraplegia and were combined with so-called anesthesia dolorosa, loss of reflexes and urinary and trophic disturbances were found. In addition, cerebral manifestations, such as diplopia, headaches, vomiting, papilledema and blindness, were present.

A definite clinical diagnosis was not made. One of us (P. B.) suspected a diffuse involvement of the central nervous system in the form of "multiple degenerative softening" described by us,[1] or a combination of myelitis and optic neuritis which has been described as "neuromyelitis optica" and has generally been considered related to encephalitis or so-called acute multiple sclerosis.

Necropsy (Dr. S. E. Brown).—Necropsy performed ten hours after death revealed among other changes numerous nodules in the peritoneum, lungs, pleura, endocardium and pericardium, small intestines (only one nodule in the large intestines), brain and cerebellum. The nodules were from 2 to 8 mm. in size, white, irregular, hard and not encapsulated, and on section they appeared grayish white. The nodule in the dura covering the right half of the cerebellum was .5 cm. in diameter; it was friable, irregular and grayish white and a "similar tumor was found on the base of the right half of the cerebellum," while in the brain only one nodule was found in the island of Reil on the right (1 cm. in diameter). The pituitary body was somewhat enlarged, and in the stalk was a white tumor 8 mm. in diameter.

In the cauda equina, a soft grayish-red tumor, which extended from the twelfth thoracic vertebra to the upper border of the sacrum, was present. The tumor entirely filled the bony canal, infiltrated the dura, the nerves and filum terminale and obscured all anatomic relations. A grayish-red tumor, 2 cm. in diameter, was present also beneath the skin in the midline of the back at the level of the fourth lumbar vertebra. Some of the cells of the nodules contained a deep brown pigment which either stained the cytoplasm diffusely or formed smaller and larger granules. The largest number of pigmented cells was found in the tumor masses which replaced the cauda equina and in some of the nodules of the lungs, intestines and myocardium. Little pigment was present in the dural nodules, in the brain, the stalk of the hypophysis and the areas which invaded the spinal cord. The relation of the pigmented and nonpigmented cells as well as the arrangement of the tumor cells varied greatly. In certain places, especially in the cauda, in the intestines and in some myocardial nodules, the cells were densely packed. By compression they assumed a spindle shape. On the basis of the foregoing observations, Dr. Jaffé and Dr. Brown made the following anatomic diagnosis: multiple metastases, probably

1. Hassin, G. B., and Bassoe, Peter: Multiple Degenerative Softening versus Multiple Sclerosis, Arch. Neurol. & Psychiat. **7**:613 (May) 1922.

from the cauda equina, of a melanoblastoma to the dura, brain, stalk of the hypophysis, myocardium, lungs, small intestines and subcutis; lobar pneumonia of the right lung; abscess in the apex of the right lung and emaciation.

Dr. Jaffé and Dr. Brown sent us the spinal cord and a larger portion of the brain for further microscopic study. This revealed practically the same changes, as outlined by Dr. Brown in his report to the Chicago Neurological Society.[2]

The condition may be summed up as alveolar accumulation of tumor cells around distended blood vessels, intermingled with necrotic areas and bordered by masses of cells filled with pigment. This accumulation was also occasionally gathered around the blood vessels.

Fig. 1.—Sacral cord. The roots of the cauda equina are infiltrated ring-like with tumor cells. Anteriorly, the infiltrating tumor masses invade the marginal areas of the anterior and partly of the lateral columns, obliterating the meningeal spaces. Over the posterolateral areas a white ring (*A*) is visible caused by the torn off pia. The numerous white spaces in the tumor masses are dilated blood vessels. The posterior columns show rarefied areas. Van Gieson stain.

Microscopic Examination (Dr. Hassin).—The most striking changes were in the spinal cord. Here the tumor mass previously described enveloped the lumbo-

2. Brown, S. E., and Bassoe, Peter: Specimens of Multiple Melanotic Tumors of the Nervous System, presented before the Chicago Neurological Society, March 15, 1928; Arch. Neurol. & Psychiat. **20**:1375 (Dec.) 1928.

sacral region and the cauda equina in the form of a broad dense ring (fig. 1). Nowhere were tumor nodules present in the spinal cord. In the illustration one can see the marginal area of the anterolateral columns for a short distance diffusely invaded by tumor cells which, however, did not form nodules but extended by way of the perivascular spaces. Otherwise, the cells were confined to and densely packed the subarachnoid space. The contents of the subarachnoid space — the denticulate ligaments, the anterior and posterior roots and the pia-arachnoid trabeculae — were obliterated and converted into a solid tumor mass. This appeared hemorrhagic and extremely

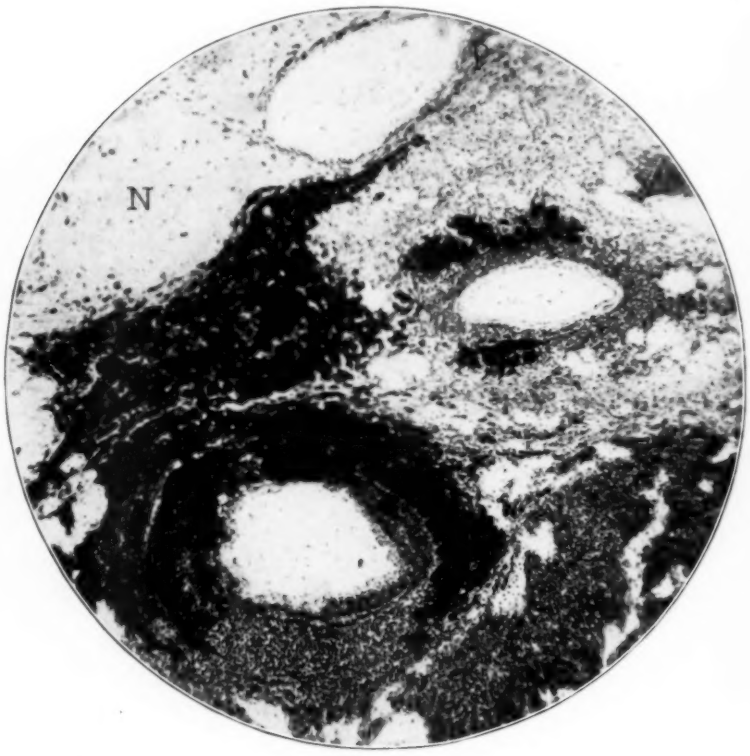

Fig. 2.—The blood vessels are patent and surrounded by dense masses of tumor cells. The black substance covering the left alveolus and portions of the perivascular spaces is melanin pigment. *P*, brownish pigment cells; *N*, necrotic area. The numerous small empty round spaces are small blood vessels. The tumor cells surrounding the blood vessels and filling the rest of the picture are shown under a higher power and with a different stain in figure 3. Toluidine blue stain.

vascular; its vessels were hyperemic, distended and surrounded by numerous concentric layers of cells (fig. 2). These formed apparently parallel rows. Blended with similar rows of the adjacent areas, they formed diffuse cellular masses, some of which appeared darker than others. They were traversed as if cut up by bundles of connective tissue, which gave the tumor an alveolar appearance. Stained with the method of Perdrau (fig. 3), each cell appeared enveloped by connective

tissue which formed a dense network covering the entire visual field. The cells were round, sometimes polygonal and always contained an abundance of homogeneous cytoplasm with a large eccentric nucleus rich in chromatin. Some cells were blended, forming a large giant cell with several nuclei. Mitotic figures were common. Fibrils were not present among or within cells, nor were they seen emanating from them.

In contrast to such masses of cells which stained well and formed the dark part of figure 1, there were large, colorless areas poorly stained in which cells could not be discerned, but only shadows of them (fig. 2*N*). Such poorly stained, color-

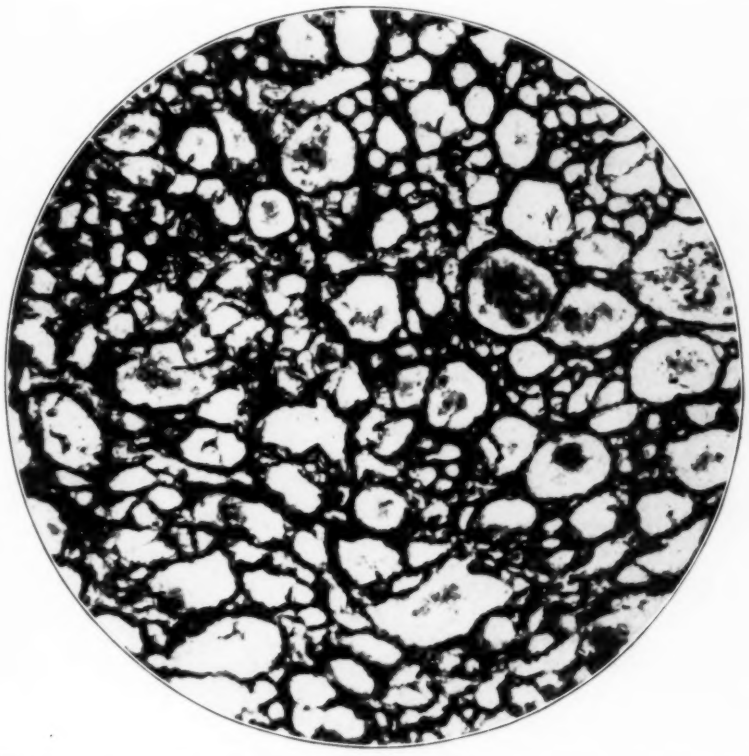

Fig. 3.—Tumor cells individually enveloped by connective tissue. Perdrau stain; ×500.

less areas were intermingled with the foregoing densely cellular portions and frequently showed an abundance of oblong cells packed with brownish pigment (fig. 2*P*). In exceptional instances the pigment formed large accumulations within the adventitial spaces of the blood vessels or filled up whole alveoli (fig. 2). As in the scattered single cells, the pigment was brownish, in the form of minute granules which densely filled the cytoplasm, crowding the nucleus to the periphery. In the photomicrograph (fig. 2), the cell contents appeared dark because of the crowded, immense masses of pigment cells. These were round, oval or slightly irregular, and without processes such as are seen in so-called chromatophore cells. While the tumor cells and the scattered pigment cells stained well with scarlet red and

sudan III, the large pigmented masses stained but slightly for lipoids and were not affected by alcohol, chloroform, xylene or acids and were bleached only slightly from hydrogen peroxide. Aside from this pigment, which evidently was melanin, there were other much darker pigment granules scattered over the hemorrhagic areas and enclosed within contiguous tumor cells. These cells gave a positive reaction to the hemosiderin test and were often intermingled with a third type of pigment granules, which varied in size and form. They were scattered irregularly, situated mostly around the blood vessels and as single droplets in tumor cells; like the melanin they were insoluble in acids, did not bleach when treated with peroxide and were not affected by alcohol or ether. These pigment granules are so-called formaldehyde pigments and do not have any pathologic significance. It should be noted that, as pointed out, large accumulations of pigment greatly resembling melanin were rare, and that the larger portion of the tumor masses were devoid of pigment and macroscopically appeared colorless.

Only rare fragments of myelin could be detected with the staining method of Weigert-Pal. Nerve fibers were not detected with any other staining methods; they all seemed to have been replaced by tumor cells. The connective tissue strands enveloping portions of the tumor exhibited distinct reactive phenomena, mainly in the form of large fibroblasts.

As noted, the tumor masses were exceedingly vascular. Their blood vessels were enormously dilated and hyperemic. In figures 1 and 2, they appear as numerous white empty spaces; their walls were occasionally hyperplastic but, as a rule, they were obliterated by immense accumulations of tumor cells which packed the adventitial spaces. In the colorless areas were large, colloid-like masses. They were also surrounded by tumor cells and were defined by Kaiser[3] as large accumulations of edematous fluid.

The changes in the subarachnoid space outlined were present around the cauda equina, as well as around the lumbosacral portions of the spinal cord. In the thoracic portion, the tumor cells became less dense; they were gathered mainly over the inner surface of the arachnoid (fig. 4), invading the posterior roots and in some instances extending to the epidural space (fig. 4*E*). In some cases the tumor cells extended by way of the arachnoid prolongations to the spinal roots, transforming some of them into a dense tumor mass. In the upper thoracic and the cervical regions, the tumor cells were confined to the perineural root spaces (fig. 5), but, as in the lumbosacral region, they did not invade the spinal cord itself.

Wherever the cells were located in small masses or as a larger tumor the pia-arachnoid exhibited reactive phenomena. In the cauda equina these could not be discerned, for the membranes were invaded and disfigured by the tumor masses. Higher up where the masses of cells were less dense, the pia-arachnoid appeared hyperplastic; the mesothelial cells were proliferated, hypertrophied and often intermingled with macrophages. Hyperplastic reactive phenomena were also present around the pial blood vessels where the tumor cells were mixed with lymphocytes, polyblasts, plasma cells and fibroblasts, some of which were of huge size.

The changes in the spinal cord were remarkable, notwithstanding the absence of tumor masses. At some levels, such as the lumbosacral cord, the white columns, especially the posterior, appeared markedly rarefied (fig. 1). They were made up of numerous vacuoles devoid of contents, broken up nerve fibers, amyloid bodies and reactive gliogenous formations such as myelophages, various types of gitter cells and cytoplasmic glia cells. In the upper thoracic and cervical regions, the white matter did not show rarefactions; instead, degenerative phenomena were

3. Kaiser, H.: Ueber primäre diffuse Sarkomatose der Leptomeningen des Gehirns und Rückenmarkes, Beitr. z. allg. Path. u. z. path. Anat. **62**:265, 1916.

present in the columns of Goll, not unlike those seen in tabes dorsalis (fig. 5). The gray matter did not show rarefaction; it showed only vascular changes. The blood vessels of both anterior and posterior horns were distended and hyperemic; their walls were hyperplastic but not infiltrated, while the ganglion and glia cells were practically normal.

Fig. 4.—The arachnoid (*Ar*) is lined by dense masses of tumor cells (*T*). In contrast to what is seen in figure 1, these fill up only a portion of the subarachnoid space. *D*, dura; *EE*, epidural tumor masses. The posterior columns show areas of rarefication. Van Gieson stain.

The nodule of the island of Reil was made up of the same cells as those found in the cauda equina. They were of the same type, were gathered around the blood vessels and were devoid of pigment (fig. 6). They did not provoke reactive phenomena on the part of the brain tissue, as it was not invaded by the tumor cells,

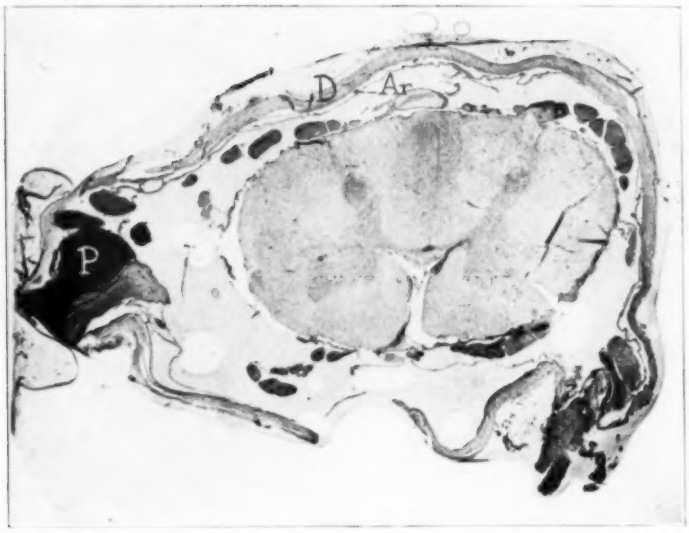

Fig. 5.—Cervical region. The left posterior root (*P*) is densely infiltrated with tumor cells forming with the lighter anterior root the radicular nerve of Nageotte. The columns of Goll are degenerated; *D*, dura mater; *Ar*, arachnoid. Toluidine blue stain.

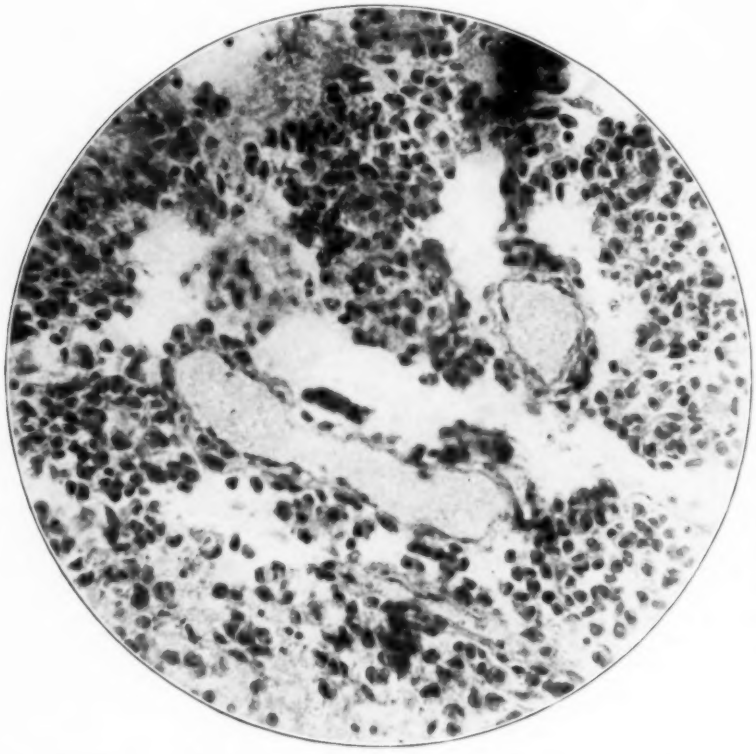

Fig. 6.—The perivascular arrangement of cells in the nodule of the island of Reil. The blood vessels are hyperemic, and the cells are devoid of pigment. Hematoxylin-eosin stain.

which were confined to the adventitial perivascular spaces. The cerebral pia, in contrast, exhibited reactive phenomena in the form of hyperemia, proliferation of the arachnoid mesothelial calls, accumulation of polyblasts and numerous other cell forms.

The cerebellum and other organs were not available for study. In his report, Dr. Brown stated that, like the tumor masses of the cauda equina, some of the nodules in the lungs, intestines and myocardium contained the "largest number of pigment cells, and that the visceral changes in general were similar to those in the brain and spinal cord. In the intestines the tumor started in the submucosa and invaded the mucosa which became necrotic."

Summary of the Microscopic Observations.—The changes observed microscopically were: tumor of the spinal subarachnoid space, extending cephalad and confined to the adventitial spaces of the blood vessels; accumulation of melanin pigment in some parts of the tumor and of lipoids in the majority of the tumor cells; absence of tumor nodules in the parenchyma of the spinal cord and their presence in the cerebellum and brain, and rarefaction and degenerative changes of the white substance of the spinal cord with reactive phenomena in the glia and meninges.

COMMENT

The tumor masses were mainly in the region of the cauda equina, which was entirely destroyed and in which the changes were at their maximum. From there the masses extended cephalad as well as along the perineural root spaces, and in some instances also invaded the epidural space. In general, the tumor cells spread along pathways normally traversed by the cerebrospinal fluid, thus pointing out, as it were, the route it followed in its circulation through the subarachnoid space and its appendages. The tumor cells in the cervical portions of the spinal cord undoubtedly came from the caudal region where they were of old standing, as evidenced by areas of necrosis, pigmentation, hemorrhages and the huge masses of tumor cell bodies. Though they formed a dense ring around the larger portion of the spinal cord, this was not disfigured or compressed, but exhibited degenerative changes similar to those seen in tabes.

The subarachnoid tumor masses were associated with such in the cerebellum, brain, pituitary body and some viscera (lungs, intestines). Generally speaking, they may be considered a manifestation of a systemic invasion by some malignant tumor. It was not possible to determine the real nature of the tumor and its exact place of origin. Long ago, Schlagenhaufer [4] called attention to the fact that in the presence of a diffuse tumor in both the meninges and parenchyma, neuropathologists are inclined to consider the primary seat of the tumor to be in the

4. Schlagenhaufer, F.: Casuistische Beiträge zur pathologischen Anatomie des Rückenmarks, Obersteiner's Arbeiten, 1900, vol. 7, p. 208.

meninges. From here they think it secondarily invades the brain or the cord. Such a view has been advocated even by the most recent investigators (Matzdorf,[5] for instance). Like Rindfleisch,[6] Schlagenhaufer [4] justly considered the reverse to be true, for, thickly as the pia may be infiltrated, the infiltration does not result in the formation of nodules within the brain or the spinal cord. Schlagenhaufer stated that in every case in which a diffuse meningeal infiltration is associated with formation of large nodules elsewhere in the central nervous system or outside it, it is secondary, that is, is the result and not the cause of such a condition. A number of observations sustain such a view. In Virchow's [7] case, for instance, the cerebral parenchyma was not involved; yet the tumor (melanosarcoma) was diffuse over the base of the brain, chiasm, pons, cerebellum and medulla. The same was true in the cases of Westphal,[8] Stoerk,[9] Minelli,[10] Boit,[11] Thorel [12] and others. On the other hand, a sarcoma of the spinal cord as a rule causes a meningeal involvement, as, for instance, in the cases of Spiller,[13] Pick,[14] Hirschberg [15] and evidently also in the cases of Matzdorf,[5] Ehmark and Jacobowsky [16] and others. The reason for a meningeal lesion follow-

5. Matzdorf, P.: Eine diffuse Geschwulst der weichen Hirnhäute, Ztschr. f. d. ges. Neurol. u. Psychiat. **86**:333, 1923; Beiträge zur Kenntniss diffuser Hirnhautgeschwülste mit besonderer Berücksichtigung der Melanome, ibid. **81**:263, 1923.

6. Rindfleisch, W.: Ueber diffuse Sarkomatose der weichen Hirn- und Rückenmarkshäute mit charakteristischen Veränderungen der Cerebrospinalflüssigkeit, Deusche Ztschr. f. Nervenh. **26**:135, 1904.

7. Virchow, R.: Pigment und diffuse Melanose der Arachnoides, Virchows Arch. f. path. Anat. **16**:80, 1859; Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1865, vol. 2, p. 119.

8. Westphal, A.: Ueber multiple Sarkomatose der Gehirn- und der Rückenmarkshäute, Arch. f. Psychiat. **26**:770, 1894.

9. Stoerk, O.: Melano-sarkomatosis piae Matris, Wien. klin. Wchnschr. **17**: 184 (Feb. 5) 1904.

10. Minelli, D.: Primärer melanotischer Gehirntumor, Virchows Arch. f. path. Anat. **183**:129, 1906.

11. Boit, H.: Ein Fall von Chromatophoroma durae Matris spinalis, Frankfurt. Ztschr. f. Path. **1**:248, 1907.

12. Thorel, C.: Ein Fall von primärer melanotischen Sarkom der Rückenmarkmeningen, München. med. Wchnschr. **54**:725 (April 9) 1907.

13. Spiller, W., and Henderson: A Report of Two Cases of Multiple Sarcomatosis of the Central Nervous System and of one Case of Intramedullary Primary Sarcoma of the Spinal Cord, Am. J. M. Sc. **126**:10, 1904.

14. Pick, L.: Einige Rückenmarkstumoren insbesondere ueber eine primäre melanotische Geschwulst (Chromatophorom) des Rückenmarks, Berl. klin. Wchnschr. **43**:884 (June 25) 1906.

15. Hirschberg, A.: Chromataphoroma Medullae spinalis des Zentralnervensystems, Virchows Arch. f. path. Anat. **186**:229, 1906.

16. Ehmark, E., and Jacobowsky, B.: Ein Fall von meningealem Melanom mit reflectorischer Pupillenstarre, Upsala Läkaref. Förh. **31**:565, 1926.

ing a tumor of the spinal cord is, as shown elsewhere,[17] due to the fact that as the tissue fluids of the brain and cord flow toward the subarachnoid space they discharge into the latter tumor cells or their catabolic products. The primary seat is usually in the cerebellum, as shown by numerous observers (Ollivier,[18] Weaver,[19] Schlagenhaufer,[4] Schopper,[20] Spiller[13] (case 2) Hamill and Rothstein,[21] Jacob,[22] Connor and Cushing[23] and others). Schlesinger[24] long ago demonstrated that a cerebellar tumor was much in evidence (ausgesprochen) in nine of fourteen cases of sarcoma of the brain and the meninges, while in a case (alveolar sarcoma) reported by one of us (P. B.),[25] in addition to a diffuse sarcoma of the spinal pia, a definite, larger tumor existed in the posterior cranial fossa. It was noted in this instance that the only large tumor rested on the cerebellum and had the structure of a sarcoma arising from the meninges. Its size and more mature structure marked it as antedating the much more cellular diffuse cord tumor; that is, the latter was secondary to a primary cerebellar tumor.

The cerebellum was affected also in the case under discussion. Unfortunately, it could not be studied in detail, since it was preserved as a museum specimen. Permission was not given to remove the eyes for study and to ascertain whether the choroid of the eye was or was not the cause of the metastases into the central nervous system and the various viscera. One should bear in mind the fact that diffuse meningeal involvement, as described here, may occur without the invasion of the parenchyma or the visceral organs, that is, it may remain a strictly local lesion. In other cases, either because of longer standing or for

17. Hassin, George B.: Effect of Organic Brain and Spinal Cord Changes on Subarachnoid Space, Choroid Plexus and Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **14**:468 (Oct.) 1925.

18. Ollivier d'Angers, C. P.: Traité des maladies de la moelle épinière, Paris, Meguignon, 1837, vol. 2, p. 490.

19. Weaver, G.: Diffuse Sarcoma of Cerebral and Spinal Pia Mater, J. Exper. Med. **3**:669, 1898.

20. Schopper, K. J.: Ueber primäre Melanosarkomatose der Pia Mater, Frankfurt. Ztschr. f. Path. **13**:77, 1913.

21. Hamill, R., and Rothstein, T.: A Case of Melanosis of the Central Nervous System, Tr. Chicago Path. Soc. **7**:266, 1906.

22. Jakob, A.: Kasuistischer Beitrag zur Lehre von den Kleinhirnbrückenwinkeltumoren und von der diffusen Sarkomatose der Meningen des Zentralnervensystems, Ztschr. f. d. ges. Neurol. u. Psychiat. **3**:249, 1910.

23. Connor, C., and Cushing, H.: Diffuse Tumors of the Leptomeninges; Two Cases in which the Process was Revealed Only by the Microscope, Arch. Path. **3**:374 (March) 1927.

24. Schlesinger, H.: Beiträge zur Klinik der Rückenmarks- und Wirbeltumoren, Jena, G. Fischer, 1898.

25. Bassoe, P., and Schields, C. L.: Diffuse Sarcoma of the Pia Enveloping Entire Cord, J. Nerv. & Ment. Dis. **44**:385, 1916.

other reasons, the spinal meningeal lesions become complicated by metastases to other organs of the body. Attempts, however, have not been made to explain how the metastases were carried in such cases from the meninges to the viscera, such as the lungs and intestines, for instance. The only ways, of course, are the lymphatics and the blood stream. In the present case the metastases to the various portions of the central nervous system were evidently carried along the adventitial spaces, as these were intensely infiltrated with the tumor cells, while the blood vessels themselves were patent, distended, hyperemic and did not contain tumor cells. The metastases most likely originated in the tumor of the cerebellum. From here it invaded the cerebral and later, of course, the spinal subarachnoid spaces, where it involved the meninges, spinal roots and especially the cauda equina. The floating tumor cells carried by the circulating cerebrospinal fluid escaped with the latter by way of the perineural root spaces (fig. 5) into the general circulation, and thus set up lesions elsewhere in the body.

The real nature of the tumor in question was also obscure. Conditions like that in the case here recorded have been termed variously perithelioma (Lissauer,[26] Nonne,[27] Guillain, Alajouanine, Mathieu and Bertrand[28]), cylindroma (Cramer[29]), alveolar sarcoma (Richter,[30] Schulz,[31] Fraenkel and Benda,[32] Bassoe[25]), diffuse sarcoma of the pia (Lenz,[33] Kaiser,[3] Andrè-Thomas and Jumentié[34]), encephaloid sarcoma (Caupland and Pasteur[35]), endothelioma sarcomatodes, angiosarcoma

26. Lissauer, Max: Ein Peritheliom der Pia Mater spinalis, Centralbl. f. allg. Pathol. u. path. Anat. **22**:49, 1911.

27. Nonne: Ueber diffuse Sarkomatose der Pia Mater des ganzen Centralnervensystems, Deutsche Ztschr. f. Nervenh. **21**:396, 1902.

28. Guillain, G.; Alajouanine, T.; Mathieu, P., and Bertrand, I.: Sarcome périthelial de la queue de cheval avec xanthochromie du liquide céphalo-rachidien au dessus de la tumeur; localization par le lipiodol, Rev. neurol. **1**:513 (May) 1924.

29. Cramer, E.: Ueber multiple Angiosarcome der Pia Mater spinalis mit hyaliner Degeneration, Inaug. Diss., Marburg, 1888; abstr., Schmidt's Jahrb. **223**: 129, 1889.

30. Richter, J.: Ueber einen Fall von multiplem Sarkom der inneren Meningen, Prag. med. Wchnschr. **11**:213, 1886.

31. Schulz, R.: Neuropathologische Mittheilungen. Case IV: Primäres Sarcom der Pia Mater des Rückenmarks in seiner ganzen Länge, Arch. f. Psychiat. **16**:592, 1885.

32. Fraenkel, A., and Benda: Zur Lehre von den Geschwülsten der Rückenmarkshäute, Deutsche med. Wchnschr. **24**:442, 457 and 476, 1898.

33. Lenz, G.: Ein Fall von diffus ausgebreiteter Sarkombildung der Pia Mater spinalis, Beitr. z. path. Anat. u. z. allg. Path. **19**:663, 1896.

34. Andrè-Thomas and Jumentié: Un cas de sarcomatose meningée diffuse, Rev. neurol. **1**:345, 1924.

35. Caupland, S., and Pasteur, W.: Two Cases of Diffuse Sarcoma of the Spinal Cord (Case 2), Tr. Path. Soc. London **38**:26, 1887.

(Busch [36]), perithelial sarcoma, perivascular endothelioma, melanosarcoma (Stoerk,[9] Thorel [12]), melanoma, melanocytoma, melanocytoblastoma, melanoblastoma, gliosarcoma and others. Fischer [37] called it glioma sarcomatodes, while in other cases in which there was diffuse involvement of the subarachnoid space the condition has been described as secondary gliomatosis of the leptomeninges (Pels Leusden,[38] Grund,[39] Spiller,[40] Firor and Ford,[41] Brannan,[42] Connor and Cushing [23]). Mallory [43] pointed out the possibility of gliomas extending into and invading tissues just like any other malignant new growths. Grund [39] stated that in twenty-nine cases of sarcoma of the meninges and the central nervous system in which diagnoses of spindle cell sarcoma, alveolar sarcoma, angiosarcoma and endothelioma were made, the condition was caused by glia tumors, while, according to Shuberth,[44] a diffuse glioma is a diffuse metatasis of a sarcomatous glioma which reached the pia or the spinal cord. Without going into a detailed discussion of the various views advocated as to the type described, we merely wish to call attention to the chaotic state of this phase of pathologic conditions in man.

In the case under discussion, the condition was still more complicated because of the presence of pigment in some portions of the tumor mass. Limited as the pigment was in our case, its presence should suggest the possibility of a pigment tumor, which may be termed melanoma or melanoblastoma. Virchow,[7] Dobbertin [45] and others termed such a tumor melanosarcoma. Ribbert [46] proposed to call it

36. Busch, C.: Ein Fall von ausgebreiteter Sarkomatose der weichen Häute des Centralnervensystems, Deutsche Ztschr. f. Nervenh. **9**:114, 1897.

37. Fischer, O.: Ueber einen selten mächtig entwickeltes Glioma sarkomatodes des Rückenmarks, Ztschr. f. Heilk. **22**:344, 1901.

38. Pels Leusden: Ueber einen eigenthümlichen Fall von Gliom des Rückenmarks mit Uebergreifen auf die weichen Häute des Rückenmarks und Gehirns, Beitr. z. path. Anat. u. z. allg. Path. **23**:68, 1898.

39. Grund, G.: Ueber die diffuse Ausbreitung von malignen Tumoren, insbesondere Gliosarkom in den Leptomeningen, Deutsche Ztschr. f. Nervenh. **31**:283, 1906.

40. Spiller, W.: Gliomatosis of the Pia and Metastasis of Glioma, J. Nerv. & Ment. Dis. **34**:297, 1907.

41. Firor, W. M., and Ford, F. R.: Gliomatosis of the Leptomeninges, Bull. Johns Hopkins Hosp. **35**:108, 1924.

42. Brannan, D.: Secondary Gliomatosis of the Leptomeninges, Am. J. Path. **2**:123, 1926.

43. Mallory, F. B.: The Results of Application of Special Histological Methods to the Study of Tumors, J. Exper. Med. **10**:575, 1908.

44. Shuberth, O.: Ueber diffuse Sarkomatose und Gliomatose in den Meningen des Zentralnervensystems, Deutsche Ztschr. f. Nervenh. **93**:34, 1926.

45. Dobbertin: Beitrag zur Casuistik der Geschwülste, Beitr. z. path. Anat. u. z. allg. Path. **28**:42, 1900.

46. Ribbert, H.: Ueber das Melanosarkom, Beitr. z. path. Anat. u. z. allg. Path. **21**:470, 1897.

chromatophoroma, though he preferred to retain the name of melanoma instead of melanosarcoma. However, chromatophoroma seemed to have gained favor, and pigmented tumors like the one here recorded have been termed chromatophoroma (Boit,[11] Pick,[14] Hirschberg,[15] Koelichen[47] and Esser[48]). Wieting and Hamdi[49] differentiated between the two types of pigmented tumors known as chromatophoroma and melanoblastoma: the former is made up of cells which secondarily "take in pigment," the latter of cells which produce pigment (melanoblasts). These are epithelial cells, while chromatophores are connective tissue or mesenchymal cells. It follows that melanoblastomas are practically carcinomas. Yet in the majority of cases in which the condition bore a close similarity to that in the case here reported, the diagnosis (probably wrong) was sarcoma, melanosarcoma or melanosarcomatosis (Pol[50]).

CHANGES IN THE SPINAL CORD

Whether the condition is carcinoma or sarcoma, such cases are of great scientific interest. They demonstrate what happens to some portions of the spinal cord when the subarachnoid space is obliterated at certain levels. The obliteration was responsible for the fact that the spinal puncture resulted in a dry tap, even though the needle was felt to be in the subarachnoid space. The point of the needle probably carried tumor cells which might have been discovered by a microscopic examination. Such an examination should be undertaken in the presence of so-called dry punctures occurring in obscure disseminated lesions of the central nervous system. It is the only method by which such lesions can be diagnosed when they are caused by tumors.

The theoretical interest is even greater. Aside from the fact that the floating cells are passively carried by the circulating fluid along its normal channels of circulation, such subarachnoid obstructions also show their effect on the tissues of the spinal cord. The effect is the same as in the experiments of Kahler and Pick, Schmaus and others in which the subarachnoid space was obliterated at some levels by foreign bodies. This has been fully discussed elsewhere.[51] The effect on the spinal cord

47. Koelichen, J.: Chromatophoroma Medullae spinalis, Ztschr. f. d. ges. Neurol. u. Psychiat. **31**:174, 1916.

48. Esser: Ueber eine seltene Rückenmarkgeschwulst (Chromatoforom), Deutsche Ztschr. f. Nervenh. **32**:118, 1907.

49. Wieting and Hamdi: Ueber die Physiologie und Pathologie der Melaninpigmentierung und den epithelialen Ursprung der Melanoblastome, Beitr. z. path. Anat. u. z. allg. Path. **42**:23, 1907.

50. Pol, R.: Zur Kenntniss der Melanose und der melanotischen Geschwülste im Zentralnervensystem, Beitr. z. path. Anat. u. z. allg. Path., supp. **7**:736, 1905.

51. Hassin, G. B.: Circumscribed Suppurative (Nontuberculous) Peripachymeningitis, Arch. Neurol. & Psychiat. **20**:110 (July) 1928.

is also similar to that in tabes dorsalis.[52] Analyzing the sections of the spinal cord at its various levels, one is struck by the degenerative changes, especially of the posterior columns. Mainly subacute and associated with immense obstruction of the subarachnoid and perineural root spaces, including the area of Nageotte (fig. 5), the conditions in the present case were practically analogous to those in tabes. In tabes, however, the changes in the subarachnoid space and the spinal roots are not so evident, because they are chronic. Another difference is that in this case the obstruction of some pathways, such as the radicular nerve of Nageotte, occurred from within, that is, from the subarachnoid space, and was caused by tumor cells; in tabes, as shown elsewhere,[52] the obstruction originates from without the epidural space and is caused by proliferated cells of the arachnoid. The effect on the cord is the same in both instances—defective draining of its tissue fluids, their stagnation, consequent rarefaction (so well seen in this case) and ultimate degeneration, so typical of tabes.

CONCLUSIONS

1. Massive subarachnoid infiltrations with tumor cells as a rule do not invade the spinal cord or cerebral parenchyma, but the reverse is true.

2. The effect of such obstructions of the subarachnoid space on the spinal cord is in the form of stagnation of the tissue fluids and rarefaction and ultimate degeneration of the spinal cord.

3. The mechanism of the lesion of the spinal cord is the same as that which obtains in tabes, thus supporting the view that tabetic degeneration of the cord is the result of deficient drainage of tissue fluids of the cord and obstruction of the so-called area of Nageotte.

4. So-called dry taps should suggest the possibility of obstruction of the subarachnoid space.

5. In cases of disseminated lesions of the cerebrospinal system of obscure origin, examination of the withdrawn needle for possible adherent tumor cells may be an aid in diagnosis.

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52. Hassin, G. B.: Tabes Dorsalis: Pathology and Pathogenesis; A Preliminary Report, Arch. Neurol. & Psychiat. **21**:311 (Feb.) 1929.

THE CEREBRAL CIRCULATION

VII. CHANGES IN CEREBRAL CAPILLARY BED FOLLOWING CERVICAL SYMPATHECTOMY *

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In a recent review [1] of the cerebral vasomotor phenomena, an explanation for the conflict of opinion concerning the existence of vasomotor control was presented. The discrepancy was attributed to the inadequate methods frequently employed, and especially to the absence of a quantitative method for directly measuring brain vessels. In this article, Forbes and one of us (H. G. W.) applied a method which was quantitative and direct for the measurement of cerebral vessels. The conclusion was reached that arteries of the brain responded by a change in diameter to stimulation of vasomotor nerves. If one accepts this experimental evidence that the nerves to the cerebral vessels have a vasomotor function, it is not unreasonable to expect that these nerves partially influence the tone of the capillaries as well as the arteries, even though the major regulation of the diameter of the brain vessels is chemical.[2]

The observations of Forbes and one of us (H. G. W.),[1] although they concern primarily the changes occurring in the arteries and arterioles, are probably indicative of what takes place simultaneously in the capillaries. Alterations in the diameter of the arteries were accompanied by a like change in cerebrospinal fluid pressure. The cerebrospinal fluid pressure was diminished following constriction of the brain vessels and was increased following dilatation. These changes were prompt and relatively short lived, and suggest that alterations in the volume of the capillary bed have taken place.

In order to answer the question of capillary behavior, it is necessary to observe directly those in the brain substance. Few if any capillaries occur in the pia mater, and, therefore, only inferences concerning

* Submitted for publication, Aug. 31, 1928.

* From the Department of Neuropathology, Harvard University Medical School.

1. Forbes, H. S., and Wolff, H. G.: Cerebral Circulation: Vasomotor Control of Cerebral Vessels, Arch. Neurol. & Psychiat. **19**:1057 (June) 1928.

2. Wolff, H. G., and Lennox, W. G.: To be published.

their response to vasomotor influences can be made by the methods referred to previously.[1] Further, all these former experiments were made over a period of only a few hours. It is of importance to determine whether such changes in capillary tone, such as might occur, could persist for longer periods.

Orr and Sturrock [3] cut the cervical sympathetic nerve on one side in animals and after varying lengths of time examined the brain vessels, without actually measuring them. They reported dilatation of the arteries, veins and minute vessels on the side operated on. Since this method depends somewhat on the amount of blood in the brain when the animal dies, it is not especially reliable.

In the present study, cats were chosen as experimental animals. The left cervical sympathetic nerve in each animal was aseptically exposed under ether anesthesia, cut and approximately 2 cm. removed. An increase in temperature of the ear and a constriction of the pupil on the side operated on were accepted as evidence that the cervical sympathetic nerve had been cut. After a lapse of time varying from one hour to six days, the vessels of the head and brain were injected with a 2 per cent aqueous solution of Berlin blue. The method of injection, described elsewhere,[4] is briefly as follows:

After anesthetization with ether, the thorax was rapidly opened and the heart and great vessels were exposed. The internal mammary arteries and the abdominal aorta were clamped. The pericardial sac was next opened, and an incision was made through the wall of the left ventricle. A cannula was inserted through the incision and carried past the aortic valves into the ascending aorta and ligatured. The injection mass, warmed to body temperature, was then allowed to run in at a constant pressure varying between 220 and 225 mm. of mercury.

Ludwig's injection apparatus was used. With this apparatus, the injection pressure was recorded and controlled throughout each experiment. The amount of Berlin blue injected into each animal varied between 150 and 200 cc. The injection was started while the animal was still alive, but within from five to ten seconds the animal was dead. The average time from the cutting of the skin until the cerebral vessels were filled with the injection mass was from forty to sixty seconds.

Great caution was exercised in the promptness with which the Berlin blue was injected. Every effort was made to get the stain into the capillaries and fix them before sufficient time had elapsed to permit carbon dioxide to accumulate. Occasionally, the period between the opening of the chest and the end of the injection was longer than a minute, and in these experiments the difference in the volume of the capillary bed in the side operated on as compared to the side not operated on was less striking. Even with the most rapid injection there was some error from this source, since in from forty to sixty seconds changes undoubtedly occurred which minimized the difference between the side operated on and that not operated on.

3. Orr, D., and Sturrock, A. C.: Lancet **203**:267, 1922.

4. Cobb, Stanley, and Talbott, J. H.: Proc. Am. A. Phys. & Surg. **42**:255, 1927.

Following the injection, the brain was rapidly removed and placed in 10 per cent liquor formaldehydi. After fixation a transverse section of the brain was cut through the cortex from 2 to 2.5 cm. caudal to the frontal pole. The sections were embedded in celloidin to insure uniform thickness on sectioning. Five sections of the whole brain, 20 microns thick, were cut serially from each brain at a level corresponding approximately to plate XII of Winkler and Potter.[5] Here three areas were measured, one near the midline, "area 17" of Winkler and Potter, and two others more laterally, areas "1 to 3" and "13 to 16." In each section the capillaries of these three areas in the side operated on as well as the side not operated on were measured and compared. Five sets of measurements were made of each area, that is, measurements of capillary length were made in blocks of tissue, 951 by 951 by 20 microns in size. Thus, in any given area the total capillary length in twenty-five blocks of tissue was measured (five blocks in each of five sections), each block containing 1,815,000 cubic microns of brain substance, as shown in the accompanying table. The five serial sections were not measured in all the brains, but in all animals at least two serial sections were measured.

In studying the sections, a square-ruled disk micrometer was used in a Leitz ocular no. 2. The objective was a Leitz no. 6 long.

Two important sources of error in the method are shrinkage in fixation and inaccuracy of measurement due to twisting of capillaries in that section. Shrinkage gives a relative error only, while the errors in measurement are probably eliminated by the averaging of a large number of measurements.

In all, operations were performed on and injections made into ten animals. Two of these, through an error in technic (air bubbles in the injection fluid) were unevenly injected, and the experiments were therefore eliminated from the series. The remaining eight brains were measured in the manner described. The animals were classified according to the time interval between operation and injection. Injections were made into two animals after one hour, two animals after twenty-four hours and four animals after six days.

Conclusions were based primarily on the careful analysis of the two six-day animals included in the table. Because of the fewer number of sections studied in the other six, the variations in measurements were much greater, but the total averages for the series indicated that there was a greater amount of injection mass on the side on which operation was performed.

The increase in every instance was greatest in area 17, less prominent in areas 1 to 3 and practically negligible or insignificant in areas 13 to 16. The location of these areas is given by Winkler and Potter.[5]

When one compares the side operated on with the side not operated on, the average percentage difference in the eight animals was as follows: Area 17 showed an increase of 49 per cent in the length of the injected capillary; areas 1 to 3, 30 per cent; areas 13 to 16, 11 per cent.

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5. Winkler, C., and Potter, A.: An Anatomical Guide to Experimental Research on the Cat's Brain, Amsterdam, W. Versluys, 1914, p. 102.

The accompanying table, showing the total measurements for the two of the eight cats, demonstrates the great variation between individual measurements and the need of a series before conclusions can be justly derived.

The great difficulty with any postmortem study of brain vessels is the significant change in diameter of the vessels that takes place just

Capillary Measurements in Two Cats

	Area 17					Areas 1 to 3					Areas 13 to 16				
	I	II	III	IV	V	I	II	III	IV	V	I	II	III	IV	V
Cat Number One															
Side Not Operated On															
1	550	1146	1542	1113	1080	1890	1209	1212	1347	1285	740	1212	918	1140	1182
2	1140	734	1692	1407	1500	1530	1176	1080	1410	1575	761	1232	993	1089	1248
3	1110	764	2070	1161	1590	1488	1440	1050	1500	1584	885	1530	1083	1254	1194
4	1050	861	1761	1860	1110	1635	1314	1776	1245	1070	1206	825	1110	1374	1170
5	680	981	1836	930	840	1506	1074	1374	1587	1320	1176	1200	1230	1131	1005
Average	906	897	1780	1294	1224	1602	1242	1298	1218	1367	953	1199	1066	1196	1159
Side Operated On															
1	1923	773	1674	2397	2556	1290	1206	1734	993	1620	963	1185	842	1554	918
2	2085	1551	1974	2298	2505	1644	1461	1854	984	1590	957	1005	907	1626	975
3	1722	1320	2205	2184	2460	1239	1935	2043	921	1410	981	1218	971	1128	1218
4	2184	1152	2331	1920	1395	1272	1938	1746	1620	1530	1002	1176	608	1143	989
5	1296	1452	1935	2010	2625	1350	2154	2292	1224	1590	677	1146	983	1050	1158
Average	1842	1449	2024	2162	2308	1359	1739	1934	1171	1528	916	1165	886	1300	1051
Cat Number Two															
Side Not Operated On															
1	1264	1305	1230	915	1425	1005	902	975	993	1206	1410	909	873	993	975
2	1207	1098	1206	707	984	945	1188	1192	1143	1077	1317	1722	776	942	879
3	1159	867	1008	782	1065	1200	999	1216	848	1086	1304	1143	824	906	842
4	671	788	1002	909	515	957	1230	957	1161	1026	1537	933	945	981	632
5	439	794	1284	882	1149	1158	1548	851	867	1674	1394	967	854	698	921
Average	944	970	1158	839	1027	1053	1185	1038	1002	1214	1392	1134	853	884	849
Side Operated On															
1	1392	1722	1375	1787	2190	1038	1331	1598	1427	1339	1042	1500	883	1535	1883
2	1159	1383	1917	1065	1713	1302	1136	1246	1307	1231	904	1308	1550	1226	1021
3	1119	1476	1305	903	1584	1530	1607	1269	1511	1418	1237	1488	817	1589	1878
4	1065	1500	1455	1023	1656	1350	1259	1154	1586	1056	1164	1592	1475	1469	1443
5	1379	1752	1512	1143	1317	1476	1217	1497	1766	1276	1002	1623	1409	1029	1476
Average	1222	1566	1537	1184	1692	1339	1310	1353	1519	1260	1082	1502	1626	1372	1540

Average Percentage Increases in Length of Injected Capillary of Side Operated On Over Side Not Operated On

	Cat Number One	Cat Number Two
Area 17	60 per cent	46 per cent
Areas 1 to 3	14 per cent	24 per cent
Areas 13 to 16	—4 per cent	39 per cent

* Figures given are for length in microns of injected capillary per 1,815,000 cubic microns of brain substance. Arabic numbers on the left margin indicate block of tissue; Roman numerals at the top indicate serial sections.

before death. Previous observations have indicated that vasodilatation accompanies asphyxia or any increase in the carbon dioxide content of the blood.[6] Therefore, if there is any difference between the capillaries or arteries of the brain on the two sides of the head as the result of the operation, it may be readily overcome or at least minimized by the effect of even slight asphyxia.

6. Forbes and Wolff (footnote 1). Wolff and Lennox (footnote 2).

The variation of response in the different "areas" calls for comment. Stohr [7] inferred from his investigations that there are several routes for cerebral vasomotor nerves at the base of the brain. All the brain vessels are not necessarily supplied by any one nerve, and any one vessel may in fact be supplied from several sources. Therefore, after unilateral section of the cervical sympathetic nerve, it is unlikely that all parts of the brain on that side will be equally affected, or even that any vessel will be permanently or seriously altered. It would seem reasonable to assume that one portion would be most affected, an intermediate portion less affected, and other portions perhaps not affected at all. This is in agreement with our observations.

When injections were made with Berlin blue, pressures greater than the systemic arterial were applied and some passive dilatation might therefore follow. This again would tend to minimize any difference in injection of the capillaries on the two sides.

If, in spite of these three leveling factors, there is still a definite and measurable difference in the capillary beds of the two sides of the brain, it is significant and justifies the conclusion that the capillaries as well as the large vessels are affected by vasomotor mechanisms.

SUMMARY

Cutting one cervical sympathetic nerve causes an increase in the capillary bed of the homolateral cerebral hemisphere.

7. Stohr, P.: Ztschr. f. Anat. u. Entwicklungs Gesellsch. **63**:562, 1922.

SYMPTOMATOLOGY OF TUMOR OF THE FRONTAL LOBE *

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The time-honored custom of subdividing the brain into lobes for physiologic study is still in vogue, notwithstanding the work of Brodmann, who showed that the homology of regions of the brain in man and animal does not follow the topography marked out by the sulci. There can be little doubt that in the future the study of the physiology of the brain according to lobes will be superseded by a more scientific modus. The frontal lobe is a large area of the brain; it is not a single mechanism with a single function. Hence, the tendency in neurology to formulate "syndromes," a group of symptoms all present or absent in a certain disease, is of questionable value here. To draw conclusions concerning the physiology and physiologic pathology of this region of the brain from symptoms observed when tumors involve the frontal lobe is fallacious, since the clinical picture is constantly complicated by neighborhood and general symptoms.

In a large series of cases of tumor of the frontal lobe, one encounters singular instances in which a distinction from subtentorial tumor is most difficult. As Cushing [1] pointedly remarked, "It is, of course, one of the traditions of neurosurgery that one may easily mistake a frontal for a cerebellar case or vice versa." However, in the long run, tumor of the frontal lobe is frequently accompanied by a train of symptoms that, when carefully studied and intelligently interpreted, allows a localization.

The abundant literature on the symptomatology of lesions of the frontal lobe lends ample proof that it is erroneous to expect the presence or the absence of the same group of symptoms in every instance of tumor of the frontal lobe. Much depends on the actual site of the lesion and on its extent. The lack of realization of this fact is responsible for the discrepancy between the opinions of authors as to the various "classical" symptoms and signs of lesions of the frontal lobe. It is obvious that only by a careful scrutiny and study of cases of frontal lobe tumor, with examination of the lesions as to their position, extent and relations to the neighboring areas of the brain, is one able

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1. Cushing, Harvey: The Field Defects Produced by Temporal Lobe Tumours, Brain **44**:395, 1921.

to form an opinion as to the relative frequency and diagnostic importance of the various symptoms and signs of these lesions.

I purpose to analyze the symptomatology observed in a series of thirty cases of tumor of the frontal lobe at the National Hospital, Queen Square, London. All these cases were under observation for a sufficiently long time to win credence for the results of the repeated examinations that were made. In every instance, a postmortem examination permitted a complete study of the topography of the tumor, its extent and its relation to the surrounding areas of the brain. The fact that all the cases of this series were observed in the same institution with uniform methods of examination adds to the reliability of the observations.

The usual tendency, in discussing the symptomatology of a tumor of a certain area of the brain, to omit the so-called "general" symptoms and to limit the analysis to symptoms of localizing value, will not be adhered to in the present study. It is known that the general and the focal symptoms are frequently intimately interdependent, and an exclusion of the general symptoms from the discussion would tend to reduce the value of the study.

The tumors of the frontal lobe of the present series were observed in twenty male and ten female patients. All except two of these tumors were confined to the frontal lobe, not involving, or little involving, the adjacent areas of the brain. The two exceptions were tumors that originated in the frontal lobe on one side and extended by way of the corpus callosum to the other side, which, however, was involved to a much smaller degree. Sixteen of the tumors were entirely limited to the left frontal lobe, and twelve, to the right lobe. Of the two patients with bilateral involvement, one had the larger mass of tumor in the right frontal lobe and the other, in the left lobe. The average age of the patients was 43 years, but a range of ages from 10 to 60 years was seen.

HEADACHES

Although they constitute the foremost member of the triad of symptoms of increased intracranial pressure—headaches, vomiting and papilledema—headaches have attracted but little attention in studies of the symptomatology of tumor of the frontal lobe. For purposes of localization, one should distinguish between headache and pain in the head. The first is a deep, more or less general, rather dull ache, in contrast to the sharp, more superficial pain limited to a certain area of the head.

Headache or localized pain in the head was complained of by twenty-six patients, but in only sixteen of them was this the first symptom. The time of onset of headache varied greatly; the average was five months

before admission of the patient to the hospital, the extremes being four days and two years. The course of the headache was the same as is usually observed in connection with tumors of the brain. That is, it appeared first at long intervals and lasted a brief time. Later on, headaches appeared at shorter intervals, and in bouts, which lasted one or more days, followed by remissions in which the patient was entirely free from them. Finally, in the advanced stage, the headaches became a constant symptom. Not infrequently, however, they disappeared completely in the last stages of the disease. The anatomic basis of the headaches in association with increased intracranial pressure is unknown, although some evidence seems to suggest that they originate within the ventricular system. The exacerbation of the headaches in the early morning, when the production of cerebrospinal fluid is at its highest, is well known. In two of the cases of this series, the headache was limited to the side opposite that of the tumor and, interestingly enough, in both these cases there was an enormous unilateral hydrocephalus on the side of the headache.

Of a different nature is the local pain observed in tumor of the brain; in most cases, the anatomic substratum of the pain is at once evident. Local pain was observed in this series in two cases, and in both the tumor was a meningioma embedded in the frontal lobe. In one, a left frontal meningioma, the pain was confined to an area above the left eye. On postmortem examination, the tumor was found compressing the left frontal lobe, pushing it back and mesially across the middle line. On its under surface, the tumor was grooved by the greater wing of the sphenoid, pressing there on the first branch of the left fifth nerve. In the other case, a tumor of the right frontal lobe, the pain was localized at the site of the tumor, although clinically an infiltration of the skull could not be made out. It is of interest that local pain is much less frequently observed in tumors of the frontal lobe than in tumors of the temporal lobe, in which local pain is usually followed by general headache and the onset of pain precedes the appearance of headache by an average interval of five months. This difference between tumors of the temporal and tumors of the frontal lobe is easily explained by the proximity of the temporal lobe to the gasserian ganglion and its branches.

Palpation and percussion of the skull is of real value in localizing a tumor. This was pointed out by the earlier clinicians; its diagnostic value is now unjustly underestimated. In five cases of this series, palpation and percussion contributed to the lateralization of the tumor. In each of these cases, there was tenderness on palpation and percussion over the side of the tumor, and in two the percussion note was definitely duller as compared with that of the opposite side.

VOMITING

In eighteen cases of this series vomiting was a symptom. The dependence of the vomiting on the headache was seen in all but one case. Commonly, vomiting appears at a considerably later stage of the disease than headache, but when present, the attack of vomiting follows closely on the heels of a bout of headaches. Early morning vomiting, when the headache is most severe, is frequently observed. Vomiting is not a constant accompaniment of the headache, and, though it makes its appearance later in the course of the disease, it may outlast the headache, so that it is not infrequently observed in a patient who does not any longer complain of headache. The "projectile" type of vomiting was rarely observed. The diagnostic importance of "projectile" vomiting in tumor of the brain has been apparently overestimated. This type of vomiting is more suggestive of a sudden increase of intracranial pressure, as in cerebral vascular disturbances, than of a gradually increasing intracranial pressure.

EYEGROUND CHANGES

In nine patients of this series, the eyegrounds remained normal up to the time of death. In nineteen patients, edematous changes of the nerve head were seen in one or both eyes, and, in two patients, a more or less advanced primary atrophy was seen.

The value of eyeground changes in localizing tumors of the brain in general, and especially a tumor of the frontal lobe, is still under discussion. The importance of the comparative degree of swelling in each eye as a lateralizing sign was greatly emphasized by Horsley,[2] and denied by Paton [3] and others. Curiously enough, both opinions have been supported by statistics. In my paper on the symptomatology of tumor of the temporal lobe,[4] I called attention to the fact that assertions as to changes in the eyeground based on too short observation are inconclusive and occasionally misleading. The ratio between the degree of edematous change of the nerve head in one eye and that in the other does not remain the same during the entire history of the tumor. A careful digest of the records of my series of tumors of the temporal lobe as well as of the present series of tumors of the frontal lobe suggests that only a comparison of the incipient signs of swelling, blurring of the edges of the disk, and fulness and tortuosity of the veins is of importance in the lateralization of the tumor. These incipient signs seem to appear

2. Horsley, Sir Victor, in a discussion of Paton: Optic Neuritis in Cerebral Tumours, Tr. Ophth. Soc. U. Kingdom **28**:136, 1908.

3. Paton, L.: Optic Neuritis in Cerebral Tumours, Tr. Ophth. Soc. U. Kingdom **28**:121, 1908.

4. Kolodny, Anatole: Symptomatology of Tumours of the Temporal Lobe, Brain **51**:385, 1928.

first in the ipsolateral eye; this was the case in the two patients of this series whose eyeground changes came under observation at an early stage. When the tumor is far advanced, the degrees of swelling are usually identical on the two sides, while in the late stage of the disease, not uncommonly the swelling in the eye contralateral to the side of the tumor is the greater.

The most characteristic syndrome of primary optic atrophy in the ipsolateral eye and papilledema in the contralateral eye associated with tumor of the frontal lobe, as described by Gowers and later by Foster Kennedy, was not so frequently present as one would like to see it. In a series of twenty-five tumors of the frontal lobe, Sachs never saw it. Obviously, optic atrophy in the ipsolateral eye can be expected only when the tumor presses directly or indirectly through the as yet uninvolved cortex on the optic nerve. In two patients of my series, this syndrome was present in a modified form. In one, the tumor came to the surface on the mesial aspect of the left frontal lobe, extending to and reaching the basal aspect of the lobe in the region of the olfactory groove, whence it passed backward to the chiasm, although it did not involve the latter. The right eyeground remained normal throughout the patient's stay at the hospital, while the left showed primary optic atrophy. In the second case, a diffusely growing tumor of the left frontal lobe came to the surface on the basal aspect of the lobe and extended across the midline to the right. Both optic nerves and chiasm were flattened out by the tumor. The sella turcica was hollowed out and the pituitary flattened down into the bottom of the sella. Both eyes showed primary optic atrophy.

INCONTINENCE

A disturbance of sphincter control was observed in eighteen cases of this series. It is more frequently associated with tumor of the frontal lobe than with tumor of the temporal lobe. In the series of tumors of the temporal lobe already referred to, this disturbance was observed in only 20 per cent of the cases. When present, it is nearly always of the nature of a relaxation; in only one case was retention of urine observed. Usually, the incontinence is ushered in by frequent micturition, which may continue for some time; this is followed by precipitate micturition and later by complete incontinence. The bladder sphincters are first involved, and the rectal sphincters in a later stage.

The disturbance of the sphincters begins much earlier in the case of tumor of the frontal lobe than in that of tumor of the temporal lobe; in the latter case, it does not appear until a late stage of the disease. In my series of cases of tumor of the frontal lobe, the disturbance of the sphincters appeared, on the average, four months before the patient's death, the extremes being one week in one case and one and a half years

in another. Patients with tumor of the temporal lobe are usually ignorant of the incontinence, while those with tumor of the frontal lobe realize the incontinence, but have lost the sense of the impropriety of it. It is interesting to speculate on the difference in the time of the appearance of the incontinence with the two groups of tumors when taken together with the difference in the personal reactions of the two groups of patients toward it. One is tempted to look on the incontinence associated with tumor of the frontal lobe as an expression of the psychic disturbances that are observed in association with this type of tumor. A definite relationship could not be made out between the disturbance of the sphincters and the site of the tumor in the frontal lobe. However, an endothelium exerting prolonged pressure on the frontal lobe with gradual and extensive disappearance of the tissue of the frontal lobe is more frequently accompanied by disturbance of the sphincters than is an infiltrating tumor of the frontal lobe.

PSYCHIC DISTURBANCES

Psychic disturbances are not pathognomonic of tumor of the frontal lobe. They may appear in the course of a prolonged increase of intracranial pressure from any cause. However, they are notorious for their occurrence in patients with tumor of the frontal lobe. In this series, psychic disturbances of one kind or another were observed in twenty-two patients.

Lesions of the left frontal lobe did not lead more frequently to psychic disturbances than those of the right lobe, which is contrary to the opinion prevalent in the literature. Psychic disturbances of one kind or another occurred in 77 per cent of the cases of tumor on the right side and in only 70 per cent of the cases of tumor on the left side. Memory defects were observed in 48 per cent of the cases of tumor on the left and in 38 per cent of cases of tumor on the right; personality changes were observed in 61 per cent of the cases of tumor on the right, as compared with 53 per cent of the cases of tumor on the left. Memory defects are more prevalent in association with tumor of the left frontal lobe than with tumor of the right owing to the disturbances of speech that occasionally accompany tumor on the left side.

This series of thirty cases is too small to generalize from, but it is of interest to note that among the male patients psychic disturbances were considerably more frequent than among the female; they were present in 80 per cent of the males and in only 60 per cent of the females.

For the purposes of analysis, it is best to subdivide the psychic disturbances observed into four groups: (*a*) defects of memory; (*b*) changes of personality and behavior; (*c*) mental confusion, and (*d*) hypersomnia.

Defects of Memory.—Definite subjective or subjective and objective evidences of defects of memory were present in thirteen cases of this series, or in 43 per cent; similar evidences were present in 50 per cent of the cases of tumor of the temporal lobe. Deterioration of memory, however, is a considerably earlier symptom in the cases of tumor of the frontal lobe. Thus, memory defects were first noticed in the patients with tumor of the frontal lobe about six months before admission to the hospital, while in the patients with tumor of the temporal lobe, memory defects attracted attention only about two months before admission to the hospital. The memory defects concern only recent events, occurrences of years past being well retained, so that while the patient remembers the year, he forgets the day and the month. In this respect, the memory defects differ from those observed in cases of tumor of the temporal lobe, in which a complaint of loss of memory for both recent and past events is not infrequently encountered.

The memory defects are not always stationary in character; not infrequently a patient has intervals of clear memory, after which the memory defects return. These intervals usually coincide with intermissions between attacks of severe headache. This suggests that memory is a function of the brain as a whole, rather than of a specific area of the cortex.

Changes of Personality and Behavior.—The psychic disturbances which can be characterized as changes of personality may be divided into two distinct groups: psychic disturbances with changes of mood and those with an alteration of the social aspect of personality, or what is usually understood as behavior. Since the appearance of these changes is usually insidious, reliable data as regards the time of their onset could not be secured.

Changes of mood were observed in thirteen patients. According to the leading element of these changes, they can be grouped under the heads of "exaltation" and "depression." A patient with changes of the first group appears cheerful, high spirited and loquacious. This condition may reach a typical euphoric stage, as observed in eight of the thirteen patients. Along with a feeling of unusual well-being there appears a persistent tendency to facetiousness. Occasionally, the facile, facetious and cheerful patient passes into a stage of irritability and moroseness. In one of the patients of this series, the depressive stage was not preceded by euphoria, but set in from the beginning. Frequently, the euphoric or depressed patient passes gradually into a stage of "childishness" or, as the French authors call it, "puérilisme." In this stage, the patient often becomes emotionally unstable, causeless weeping alternating with irresistible laughter. This impulsive crying and laughing, Sachs believes, is most characteristic, and of great diag-

nostic value when present. I have observed similar impulsive crying and laughter on three occasions in patients with other cerebral lesions, mostly of a vascular nature.

Changes in behavior were observed in seven patients. The patient becomes self centered and extremely egotistic. He loses interest in his social functions as a member of the community. He becomes shameless about manners, clothes, expressions and morals. He shows complete loss of sexual control, the normal inhibitions of the sexual instinct being absent. One patient, aged 58, abused his wife, aged 45, despite his complete sexual impotence; another patient, aged 47, attacked his niece and his housekeeper. I am tempted to believe that the tumor in these cases completely abolished the inhibitions developed in man in the course of civilization.

Mental Confusion.—This change was observed in only three cases of this series. One patient, brought from Wales, refused to believe that he was in London; he also was confused as to the time, maintaining that it was the time of the World War. Another patient was disoriented as to both time and place; he could easily be corrected but would again lose the thread. A third patient would have constant "daydreams," as his family called it; he would "dream" about the past and the future, connecting facts with fantastic ideas. He could, however, easily be "aroused" from this stage.

Hypersomnia.—In studying prolonged sleep in patients with tumor of the brain, it is imperative that one discriminate carefully between actual prolonged sleep and the drowsiness and stupor commonly observed in patients with prolonged increase of intracranial pressure. Hypersomnia, as Purves Stewart called the prolonged sleep seen in some patients with tumor of the brain, was present in only four cases of this series, or in 13 per cent. This is a much lower incidence than was found for the series of tumors of the temporal lobe, in which hypersomnia was present in 23 per cent of the cases. In three of the patients with tumor of the frontal lobe there was a history of actual prolonged sleep in the absence of headaches or drowsiness. In one patient, persistent rubbing of the nose and yawning were the most characteristic features. The anatomic site of the tumor in these four cases does not support the opinion that prolonged sleep is observed only in association with tumor of the region of the third ventricle of the brain.

FITS

The importance of fits in the diagnosis of tumor of the frontal lobe has received little attention. An opinion has even been expressed that the only type of fits occurring with lesions of the frontal lobe is the conjugate deviation of the head and eyes to the side opposite that of the

site of the lesion. To be of localizing or lateralizing value the fits must be typical of a definite location of the discharging lesion; it must have a local beginning, or at least it must allow of a conclusion as to the lateralization of the lesion from the condition of the reflexes immediately following it. It is mainly with this point in view that fits will be discussed here.

Fits of one kind or another were a symptom in fifteen cases of this series, but only in a small number of cases were the fits of localizing or lateralizing value. Typical jacksonian fits were seen in three cases; obviously, in these cases the fits were of foremost localizing importance. The fits observed in three other cases spread too rapidly to allow of an accurate conclusion as to the location of the discharging lesion, but the more active participation in the fits of one side of the body made the fits of lateralizing significance. In five cases, motor fits were not of localizing or lateralizing significance; that is, observation of the patient by his family during the fits did not give a clue to the site of the lesion.

The incidence of sensory fits is small in patients with tumor of the frontal lobe. Only when a tumor of the frontal lobe is extensive can it provoke a sensory fit. Sensory fits occurred in two patients of this series; in one, a typical jacksonian fit was followed by pain in the contralateral lower extremity. The tumor came to the surface at the vertex just anterior to the upper end of the rolandic fissure. In the other patient, the sensory fits consisted of uncinate attacks. This patient smelled peardrops daily, beginning about one month before admission to the hospital. A large tumor involving the right frontal lobe was present, the temporal pole was pushed mesially and part of its inner edge herniated through the incisura tentorii.

In four patients, attacks of petit mal were observed. In all these, the petit mal consisted of "fainting" with loss of consciousness for a brief moment, after which the patient would immediately continue his interrupted work. Convulsions did not occur during these attacks. Usually, the attacks would come on at intervals of many months, and only in the later stages of the disease would the intervals grow shorter. One patient had had an attack of petit mal fourteen months before admission to the hospital and none thereafter. The first symptom in another patient was "a fainting spell" thirty-nine months before admission to the hospital; he was then free of symptoms for a whole year; then another "fainting spell" occurred, following which the spells returned after eight months, six months, five months and two months. Just before his admission to the hospital, these spells occurred three times a week. Headaches were the second symptom; they made their appearance fifteen months after the first fainting spell. There can be little doubt that the fainting spells were epileptic fits, although it is difficult to explain them physiologically.

Fits were not a late symptom in these patients with tumor of the frontal lobe; the average time of onset of the fits was more than seventeen months before admission to the hospital; the latest onset was two months before admission, and the earliest six years before admission. The earliest onset occurred in a patient, aged 10, with a diffuse tumor of the right frontal lobe. In this child, the fits remained the only symptom for over five years; then the patient became emotionally unstable and tried to commit suicide. Headaches and papilledema appeared only about six months before his admission to the hospital. Focal sensory fits are a late symptom of tumor of the frontal lobe. Attacks of petit mal occurred, on the average, twenty-nine months before admission to the hospital, with the longest period before admission four years.

Notwithstanding the fact that only in six cases of this series were fits of localizing or lateralizing significance, they were the first symptom of tumor of the frontal lobe in eleven cases, according to the patient or his family. The fact that the average age of the fifteen patients with fits in this series was 44 years and that the fits long remained the only symptom in eleven patients, stresses the diagnostic importance of fits appearing for the first time in patients of middle age.

MOTOR AND SENSORY DISTURBANCES

Purely anatomic considerations suggest that motor and sensory disturbances are a late symptom of tumor of the frontal lobe. These disturbances might be expected to appear only after the tumor has extended to and has encroached on the precentral area of the brain, and thus has ceased to be a tumor of the frontal lobe. The observations in the present series, however, clearly contradict these considerations; however one chooses to explain the causation of these disturbances, whether as the result of a direct encroachment by the tumor or, better, as neighborhood symptoms, the facts stress the importance of motor and sensory disturbances in the diagnosis of tumor of the frontal lobe. The motor disturbances observed in this series of cases may be subdivided into: (*a*) defects of conjugate deviation of the eyes, (*b*) facial weakness of central origin and (*c*) all other motor disturbances.

Conjugate Deviation of the Eyes.—Gordon Holmes emphasized the importance of defective conjugate deviation of the eyes to the contralateral side in the diagnosis of tumor of the frontal lobe. This deviation is a result of involvement of the frontal eye field in the region of the mesial frontal gyrus. In four cases of the present series, such a defect of conjugate deviation was seen repeatedly. In all four cases, the tumor involved directly or by pressure the frontal eye field. In three, the tumor was situated on the left side, but only in two of the three was

there an emissive speech defect. In the third, speech was normal, but the defect of conjugate deviation of the eyes to the right betrayed the seat of the tumor. In my article on the symptomatology of tumor of the temporal lobe, I emphasized the fact that a defect of conjugate deviation of the eyes is not always diagnostic of involvement of the frontal eye field, since a lesion in the region of the superior temporal gyrus may also cause such a deviation.

Facial Weakness.—Sachs,[5] in a recent paper on the symptomatology of tumor of the frontal lobe, emphasized the diagnostic value of a weakness of the lower portion of the face on the side opposite to the tumor. A contralateral central facial weakness was encountered in twenty-two cases of the present series. In several instances, this weakness was noticed only after prolonged observation. It may come and go; in one patient it was present only when the headaches were at their worst. It is usually best seen by the observer while he is in conversation with the patient, and when the latter is ignorant of being watched for such a lagging or drooping of the face in early mimetic movements. The extremely elusive, transitory nature of this weakness suggests that it is a result of pressure on the facial center. This weakness is noticed more frequently than any other pressure phenomenon, because the mimetic movements are of a much more delicate nature and may be perceived by the examiner without the patient's subjective help in the examination.

The facial weakness was not always of the nature of voluntary movements, for it was a result rather of emotional expression, the so-called mimetic facial weakness. These observations tend to support Kinnier Wilson's suggestion that there is a separate path for emotional expression running from the frontal lobe to the facial nucleus. This path is distinct from the pyramidal system and may be injured independently of it. It is of interest to note that weakness of the tongue frequently accompanies facial weakness. Usually, deviation of the tongue (when protruded) to the side opposite that of the tumor appears at a later stage of the disease. However, in three cases, deviation of the tongue was present in the absence of facial weakness; this deviation of the tongue unaccompanied by facial weakness was present throughout the lives of these patients. The importance of the deviation of the tongue in the lateralizing of these three cases is obvious.

Other Motor Disturbances.—Apart from facial weakness, motor disturbances involving the body or the extremities were present in fifteen cases. In ten of these, facial weakness accompanied the motor disturbances and in the other five facial weakness was not noticed. In three of the last five of the cases, the motor disturbances were ushered in by

5. Sachs, Ernest: Symptomatology of a Group of Frontal Lobe Lesions, Brain **50**:474, 1927.

contralateral weakness of the muscles of the tongue. In nine cases, there was a more or less complete hemiplegia of the side contralateral to the tumor. In five cases, the upper extremity alone was weak, and in one the lower extremity only was involved. The prevalence of involvement of the upper extremity was noted also in the four of the nine cases with hemiparesis; weakness of the upper extremity preceded that of the lower for some time. In the cases in which the lower extremity alone was involved, the tumor was situated high up, coming to the surface on the mesial aspect in the region of the upper end of the rolandic fissure. In the four cases in which the motor weakness appeared simultaneously in the lower and the upper extremities, the tumor came to the surface about the vertex of the frontal lobe in front of the upper end of the rolandic fissure.

The fact that deviation of the protruded tongue to the side opposite that of the lesion frequently preceded other motor disturbances, and that the involvement of the upper extremity was more frequent and more pronounced than that of the lower extremity, can be explained by the anatomic relation of the respective cortical motor centers to the frontal lobe, as well as by the more frontal course, toward the internal capsule, of the nerve fibers of the tongue and the upper extremity which originate in the motor areas of the cortex, as compared with the course of the corresponding fibers of the lower extremity.

Sensory Changes.—Sensory disturbances were observed in only two cases. In one, there was pronounced astereognosis of the contralateral upper extremity in the absence of any motor disturbances, while in the other there was definite loss of discrimination of two points in the presence of marked weakness of the entire side of the body.

Tremor.—A unilateral tremor of the extended ipsolateral upper extremity is an interesting motor disturbance occasionally seen in association with tumor of the frontal lobe. This tremor is especially well seen when the patient extends his hands in front of him with the palms facing down and the fingers separated. The tremor can be perceived easily by holding one's palm against the separated fingers of the patient's outstretched hands. It is a rapid fine vibratory tremor not unlike that in paralysis agitans, although of a smaller range and a finer rhythm. The tremor disappears during muscular rest. To perceive the tremor, the observer must examine the patient repeatedly since it frequently comes and goes.

Unilateral tremor was observed in three patients of this series. In two, a tremor was noticed in the contralateral hand, but it was slower and coarser. A slight motor weakness was associated with the tremor, so that motor weakness suggests itself as the cause of the tremor in these two cases. The rapid, fine tremor of the ipsolateral hand resembled the tremor one sees in connection with exophthalmic goiter.

The tremor was described, in 1906, by Grainger Stewart [6] as a feature of tumor of the frontal lobe. At that time, Stewart said: "I have never met with similar tremors in association with tumors situated in other regions of the brain." I had occasion to call attention to a similar tremor in association with tumor of the temporal lobe with which it is occasionally present as an ipsolateral or contralateral phenomenon. The anatomic basis of this tremor has not been illuminated by the various explanations advanced.

The Reflexes.—The deep tendon reflexes are of limited importance in the diagnosis of tumor of the frontal lobe. Changes in the reflexes were demonstrable usually after the tumor had reached an advanced stage and could be localized from other signs. Of greater value were the abdominal reflexes and the plantar responses. The result of a comparison of the abdominal reflexes of the two sides was more important than the presence or complete absence of the reflexes. The diagnostic significance of the abdominal reflexes is greater in lesions about the central and the precentral area and is of secondary value in tumor in front of or behind this area. In involvement of the pyramidal tracts the abdominal reflex disappears, although the preservation of the reflex does not exclude a lesion of the pyramidal tract.

In eight cases of this series, the abdominal reflex was of definite diagnostic importance. The abdominal reflex on the contralateral side was completely absent or slower in response and quicker in tiring out than on the ipsolateral side. In one case, the abdominal reflex was absent on the ipsolateral side; a tumor of the right frontal lobe led to a diffuse enlargement of it, with marked pressure on the vertex of the left hemisphere just in front of the left motor area.

As compared with the abdominal reflexes, the plantar response was of greater diagnostic importance. This is of special interest, since, in the case of tumor of the temporal lobe, the opposite is true. It seems that in association with tumor of the frontal lobe the appearance of an extensor plantar response takes place at an earlier stage than the disappearance of the abdominal reflex. In evaluating the plantar response, one must realize that a turning of the foot inward with raising of the knee and with slight fanning of the toes may precede the appearance of the typical extensor response as originally described by Babinski: a slow meditating response, as though the plantar irritation has to travel up the cord and back to the toes.

The contralateral plantar response was extensor in fifteen cases of this series, while in three the response remained a questionable extensor

6. Stewart, T. G.: The Diagnosis and Localization of Tumors of the Frontal Regions of the Brain, Lancet **2**:1209, 1906.

or a questionable flexor during the patients' stay at the hospital. A careful study of the evolution of the plantar response in a patient with a tumor of the frontal lobe, as based on daily examinations during a prolonged period, reveals the following successive stages: the normal flexor response becomes a doubtful flexor, then a questionable extensor and finally a definite extensor. When the plantar response is questionable flexor or questionable extensor on repeated examination, it has definite corroborative diagnostic value when taken with the other evidence at hand.

One patient of this series presented features of a reflex nature that justify a more detailed account.

A man, aged 57, was admitted to the National Hospital, under the care of Dr. Hinds Howell, with a complaint of vomiting, weakness of the left side and loss of memory, and a history of fits of the left side for two years, motor weakness of the entire left side of the body for a year and character changes for six months. The following observations are of especial interest. "When the fingers of the observer's hand, placed in the palm of the patient's right hand, are slowly withdrawn, he makes an instant grasping movement. If the withdrawal is continued, the grasp tightens to such an extent that the patient can be drawn bodily up from the bed. When withdrawal is effected, the patient's flexed fingers rapidly extend almost fully and after a pause of a second, come more slowly back into help-flexion (nachgreifen)." At the postmortem examination a spherical meningioma, measuring 7.5 by 8 cm., was found embedded in the right frontal lobe, of which only the pole and a narrow strip of the basal surface was left.

This reflex was first described by Janischewsky,[7] in 1909, who called it "réflexe saisisseur." In 1927, Adie and Critchley[8] reviewed the literature, added three more cases and named this reflex the "grasp reflex." These authors stated: "We are convinced that their presence (forced grasping and groping) in a patient with a cerebral tumor is unequivocal evidence of the situation of the tumor (in the frontal lobe on the side opposite to the hand showing the grasp reflex)." The literature dealing with this rare phenomenon affords discrepant opinions as to its explanation. It is viewed by some as a part of a syndrome mainly characterized by apraxia. Others credit lesions of the basal ganglia with its causation, but Adie and Critchley support Janischewsky in his surmise that this reflex is a result of disease of the frontal lobe. The case cited here is of interest, since the grasp reflex was present in the ipsolateral upper extremity, while the contralateral extremity showed hemiparesis.

7. Janischewsky, A.: Un cas de maladie de Parkinson avec syndrome pseudobulbaire, Rev. neurol. **18**:823, 1909.

8. Adie, W. J., and Critchley, Macdonald: Forced Grasping and Groping, Brain **50**:142, 1927.

DISTURBANCES OF GAIT AND BALANCE

The control of posture by the central nervous system is still unsolved, despite the voluminous literature. Maintenance of normal gait and balance has been ascribed, other than to the cerebellum, to the cerebrum. Apart from the occasional finding of ataxia in association with tumor of the temporal lobe, some authors speak of a "temporal lobe ataxia." In a study of the function of the frontal lobe based on observations of patients with war lesions, Feuchtwanger [9] spoke of spontaneous disturbances of balance that he had observed frequently in association with lesions of the frontal lobe. He concluded his analysis of this question by a statement: "Es ist sicher, dass die Stirnhirnrinde zu den funktionierenden Gliedern im zentralen Gleichgewichtsapparat gehoert." The doubtfulness of this statement is seen from the fact that, even among the supporters of the opinion that the frontal lobe is an organ directly controlling posture, unanimity is not reached as to the influence of lesions of the frontal lobe on balance. Oppenheim [10] said that a patient with a tumor of the frontal lobe falls to the side of the tumor, while Williams [11] reported cases in which the patient showed a tendency to fall to the contralateral side.

Of the series of patients under discussion, only two showed what one could call pseudocerebellar signs. One patient, with a tumor of the right frontal lobe revealed a tremulous right hand in the finger-to-nose test, and a right arm that did not swing normally when he was walking. The other patient, with a left frontal endothelioma, showed adiadokokinesia in the left hand. In neither of these cases was there any evidence of pressure through the tentorium on the ipsolateral cerebellar hemisphere. Thus, the incidence of disturbances of gait and balance was exceedingly small in this series of cases. While it is fallacious to draw conclusions as to the normal function of the frontal lobe from observations on patients with intracranial tumors, the scarcity of disturbances of control of posture in this series definitely speaks against the diagnostic importance of these disturbances when tumor of the frontal lobe is under consideration.

SPEECH DISTURBANCES

In evaluating the diagnostic significance of disturbances of speech in a case, one must distinguish between the disturbances that characterize true aphasia and the disturbances that are provoked by prolonged increase of intracranial pressure, by slow cerebration and by loss of

9. Feuchtwanger, Erich: Die Funktionen des Stirnhirns, Berlin, Julius Springer, 1923.

10. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, Berlin, S. Karger, 1898, p. 635.

11. Williams, E. M.: Lesions of the Frontal Lobe, M. Rec. **90**:764, 1916.

memory. The latter are characterized by hesitancy and slurring of speech in the absence of any sensory or verbal speech defects. The patient wanders off repeatedly and loses the thread of his narrative; when constantly prompted in his story, his emissive speech appears normal. As a talkative normal person loses the thread of his theme, so this patient loses the thread of his phrase; he forgets the beginning of it by the time he reaches the middle. These disturbances easily simulate true aphasia, and in the literature one occasionally finds them classed as such. Sachs found such disturbances in ten of fifteen patients with tumor of the left frontal lobe and in three of ten patients with tumor of the right frontal lobe. He classed the disturbances as aphasic, though he realized that "it may be open to question whether we are justified in calling a mere hesitancy and slowness of speech a form of aphasia." That this is a wrong course one sees from the fact that such disturbances occurred in three patients with tumor of the right frontal lobe.

The segregation of aphasic disturbances of speech into groups, according to the degree of involvement of the main components of normal speech is of doubtful value in the symptomatology of tumor of the frontal lobe. The type of the neoplasm, its extent, the condition of the ventricular system and the reaction of the adjacent tissue are the determining factors in the production of speech defects of various shades. In general, however, the speech defects produced by a tumor of the frontal lobe are characterized by disturbances of emissive speech in the presence of normal sensory speech. In the late stages, a slight aphasia of comprehension sometimes accompanies the aphasia of outgoing speech.

In the present series, aphasic speech disturbances were observed in eight patients with tumor of the left frontal lobe. The other nine patients with tumor of the left frontal lobe, as well as all thirteen patients with tumor of the right frontal lobe, were free of any aphasic symptoms. Aphasia was observed in seven of eleven male patients with tumor of the left frontal lobe and in one female patient of six patients with tumor of the left frontal lobe; i.e., in 64 per cent of the male and in 17 per cent of the female patients with tumor of the left frontal lobe. The fact that aphasia was observed in only eight of seventeen patients with tumor of the left frontal lobe speaks for the diagnostic limitation of aphasia as a symptom of tumor in this location. This conclusion tallies with the fact that in only three of these eight was aphasia the first symptom.

VISUAL DISTURBANCES

Of the three varieties of visual disturbances observed in patients with tumor of the brain—concentric constriction of the visual field, central scotomas and hemianopic defects—the first is of the least

importance in localizing the growth. It is a result of prolonged swelling of the nerve head and, as such, is merely an indirect sign of increased intracranial pressure. In my series, concentric constriction was observed in five patients; in four it was bilateral and accompanied a high grade swelling of the nerve head, but in the fifth, a patient with a tumor on the left side, it was in the left eye while the right eye was completely blind as a result of secondary optic atrophy. Ossasionally, concentric constriction is associated with a central scotoma or a hemianopic defect; in these infrequent instances, only the central scotoma or hemianopic defect is of localizing value.

The finding of the visual defects recorded here was based on perimetric examination for white color only. It is well established that quantitative perimetry is more reliable than qualitative color perimetry. The taxation of visual perception increases first of all with the decrease in the size of the test object, and only in the second place does it depend on the color of that object. Central scotomas have been described frequently as occurring in association with tumor of the frontal lobe. They were unmistakably present in two patients of my series; a large central scotoma was present in both eyes of one of them, while the ipsolateral eye of the other was completely blind at the time of admission to the hospital, the contralateral eye showing a central scotoma. In both instances, the tumor came to the surface on the basal aspect and extended backward to the region of the optic nerves and the chiasm. One patient had a tumor on the mesial aspect of the left frontal lobe reaching the inferior aspect of the lobe in the region of the olfactory tract whence it passed backward to the chiasm, pressing somewhat on the right half of the latter. Besides a bilateral central scotoma this patient also had a well expressed left homonymous defect. It was the association here of the hemianopic defect with a central scotoma that kept one from localizing the lesion in the right cerebral hemisphere.

Hemianopic visual defects were present in four patients; in two, the defects were demonstrated on perimetric examination, while in the other two the perimeter did not show a visual defect, but a test for visual inattention revealed a homonymous inattention-hemianopia. The homonymous defect in one of these cases, that referred to in the preceding paragraph, was associated with a central scotoma. In the other three cases, the contralateral homonymous visual defect at once lateralized the tumor. In these cases, the tumor came to the surface on the basal aspect of the frontal lobe, extending back to the ipsolateral optic tract and coming into close relation with it. Apparently, the homonymous visual defect was caused by pressure on the optic tract. In this series, a hemianopic defect was not caused, in any case, by pressure on the optic radiation where it passes in front of the inferior horn of the lateral ventricle. This is probably for the reason that only cases with the tumor

limited to the frontal lobe were selected for this study. The subjective visual disturbances were not of localizing significance. There was complaint of visual disturbances in only eight cases. This is in marked contrast with the observation in the series of tumors of the temporal lobe in which subjective visual disturbances were present in 50 per cent of the cases. The usual complaint was of failing vision. The average time of onset of this symptom was about three months before admission to the hospital.

HEARING, SMELL AND TASTE

Unilateral disturbances of hearing were present in only three of my thirty-eight cases of tumor of the temporal lobe. This fact is in accord with the generally held opinion that the cochlear fibers of the auditory nerves do not decussate completely. Considering this rarity of disturbances of hearing in association with tumor of the temporal lobe, one is not surprised that these disturbances are never a symptom of tumor of the frontal lobe. In none of the cases of the series under analysis was there subjective or objective evidence of disturbances of hearing that could be placed in connection with the history of the tumor.

In the case of tumor of the frontal lobe, disturbances of smell arise when the tumor presses directly or indirectly on the olfactory tract or lobule. This was observed in three cases of this series. In all three cases, anosmia was complete and bilateral, the tumor having been large enough to extend pressure across the midline. Sachs, speaking of three cases with subjective disturbances of smell in his series of twenty-five tumors of the frontal lobe, said that "in at least one of them was a definite involvement of the temporal lobe which doubtless accounted for this disturbance." I believe that anosmia hardly ever occurs associated with tumor of the temporal lobe, for it is only when the temporal lobe tumor extends forward along the orbital roof that it may encroach on the olfactory tract and cause disturbances of smell. In my series of thirty-eight cases of tumor of the temporal lobe, anosmia was not observed once, notwithstanding the fact that the olfactory center is situated in the temporal lobe. A slight depression of the acuity of smell and taste is not infrequently present as a result of prolonged increase of intracranial pressure. Complete ageusia was not observed in any of the cases of this series.

CRANIAL NERVES

Apart from the olfactory and optic nerves, only the oculomotor, trochlear and abducens nerves are to be considered in connection with tumor of the frontal lobe. Weakness of the muscles supplied by the trigeminus, the facialis and the hypoglossus is due to a central involvement of the motor area of the cortex and not to an involvement of the peripheral nerves.

The symptoms and signs of an involvement of the oculomotor and trochlear nerves may be grouped under three headings: diplopia, pupillary changes and ptosis. Diplopia was complained of by nine patients. In patients with tumor of the frontal lobe, diplopia is a transient symptom; it comes and goes. This suggests that diplopia in these cases is a neighborhood symptom, a result of pressure on the nerve by the edematous tissue adjacent to the involved area.

The pupillary changes are of two varieties: changes in the size of the pupil and changes in its shape. The changes of size are much more frequent than those of shape. In nine instances changes in size of the pupils were observed, while changes in shape accompanied the changes in size in only two patients. Myosis of the ipsolateral pupil precedes mydriasis, which appears in the later stages. Myosis may be exceedingly slight and elusive; it may remain for a day or two and then disappear completely, only to return at a later date, more marked. With further progress of the tumor, the ipsolateral pupil becomes dilated and wider than its normal fellow—myosis gives way to mydriasis. This state of the pupil remains to the end of the disease.

Ptosis was observed in only two patients. In both it was ipsolateral with the tumor and in both it appeared after the pupillary changes.

In three cases of this series there was definite evidence of a bilateral paresis of the abducens. In all three it came on late in the disease and, consequently, lacked localizing value. Sir William Gowers [12] pointed out the fallacy of relying, in the diagnosis of advanced intracranial tumor, on an abducens paralysis as a localizing sign. It is usually considered that such a paralysis is much more common in the case of subtentorial or midbrain tumor than in the case of cerebral tumor. My three cases of bilateral involvement of the abducens were associated with far advanced large tumors of the frontal lobe.

SUMMARY AND CONCLUSIONS

The frontal lobe is too large an area of the brain for lesions of it, variable in type and extent as they are, to produce any one definite train of symptoms and signs. For these reasons, I shall not attempt to formulate here a definite syndrome for tumor of the frontal lobe, but shall merely enumerate in the order of their frequency the various symptoms and signs encountered in this series of thirty tumors of the frontal lobe.

Headache, though complained of in twenty-six cases, was the first symptom in only sixteen (53 per cent). Headache was merely a general symptom of increased intracranial pressure and did not have local-

12. Gowers, Sir William: Diseases of the Nervous System, London, J. & A. Churchill, 1893, p. 180.

izing value. Local pain in the head, observed in two cases, was of definite localizing importance. Palpation was of real localizing value; tenderness on palpation of the skull overlying the tumor was noted in five cases of this series.

Vomiting was present in 60 per cent of the cases. It is a general symptom of increased intracranial pressure and does not have any localizing value.

The eye grounds in 30 per cent of the cases remained normal up to the end of the patients' lives; in 63 per cent papilledema of the nerve heads was seen in one or both eyes. The comparative degree of swelling of the nerve heads on the two sides was of some lateralizing value but only in the incipient stages of papilledema. The syndrome of primary optic atrophy in the ipsolateral eye and papilledema in the contralateral eye was present, in a modified form, in but two cases.

Incontinence was observed in 60 per cent of the cases. It was about three times as frequent as in my series of cases of tumor of the temporal lobe. While in association with tumor of the frontal lobe, disturbances of the sphincters seemed to be a result of psychic disturbances, in association with tumor of the temporal lobe, it was merely a symptom of increased intracranial pressure.

Psychic disturbances occasionally dominated the entire clinical picture, so that they alone called the patient's attention or that of his family to his condition. When an intracranial tumor had been diagnosed, certain psychic disturbances in some instances helped to localize it in one or another part of the brain. The tendency to attribute psychic disturbances to lesions of the left frontal lobe alone was not supported by clinical facts, so that psychic disturbances did not have lateralizing value. Defects of memory were present in 43 per cent of the cases. The defect of memory concerned mainly recent events, in contrast with the observation in the series of tumors of the temporal lobe, which were not infrequently accompanied by a loss of memory for both recent and past events. Changes of personality were observed in 43 per cent of the cases; the majority of the patients with these changes showed "Witzelsucht," which has long been considered characteristic of lesions of the frontal lobe. Hypersomnia and mental confusion were of diagnostic importance only when a subtentorial tumor was to be considered, with which they hardly ever occur.

Fits occurred in 50 per cent of the cases, but only in 20 per cent were they of localizing or lateralizing significance. Sensory fits were rare in association with tumor of the frontal lobe. Attacks of petit mal were seen in four patients; and in two of these, petit mal remained the only symptom for over three years, after which headaches appeared.

Motor disturbances in association with tumors of the frontal lobe were neighborhood symptoms. The most frequent disturbance was a

contralateral lower facial weakness; it was observed in over 73 per cent of the cases. In 50 per cent of the cases, there was motor weakness of one or both contralateral extremities. In 10 per cent of the cases, there was a deviation of the protruded tongue to the contralateral side, while in over 13 per cent of the cases, a definite defect of conjugate deviation of both eyes to the contralateral side was seen. In 10 per cent of the cases, a fine vibratory tremor of the ipsolateral hand was noticed. One patient showed forced grasping and groping in the ipsolateral hand when touched ("grasp reflex").

The nature of the plantar response was of more importance in the diagnosis of tumor of the frontal lobe than the condition of the abdominal reflexes. An extensor plantar response was seen in 50 per cent of the cases, and in 10 per cent more, the response remained questionable during the entire stay of the patients in the hospital. The abdominal reflexes were of diagnostic importance in only 27 per cent of the cases.

With regard to disturbances of gait and balance, these thirty cases of tumor of the frontal lobe did not support the claim of the importance of the frontal lobe in control of posture.

True aphasic disturbances of speech were seen in 47 per cent of the patients with tumor of the left frontal lobe and in none of tumor of the right frontal lobe. In only 17 per cent of these patients with tumor of the left side was aphasia the first symptom.

Subjective visual disturbances, as well as the most frequent type of objective disturbances, concentric constriction of the visual fields, were not of localizing value. Visual field defects of lateralizing importance were observed in only 20 per cent of the cases; these defects were of the hemianopic or central scotoma variety.

Disturbances of hearing, smell and taste did not have any localizing significance except in those infrequent cases when the tumor was pressing directly or indirectly on the olfactory lobule or tract.

Diplopia of a transitory nature was complained of by nine patients (30 per cent). Ptosis was observed in two patients, and oculopupillary changes were present in nine (30 per cent). In 10 per cent, there was a bilateral paresis of the abducens.

THE INCIDENCE OF GROWTH DISORDERS IN NINE HUNDRED AND TWENTY-THREE CASES OF MENTAL DISEASE *

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Irregularities in the exterior conformation of the human body have been regarded as an indication of the general constitution for a long time. In the beginning of the nineteenth century such irregularities were thought to signify that the person represented a deviation from the "normal temperament" which the old physicians had called the "temperamentum temperatum." With the rise of the conception of degeneration, following Morel, these irregularities under the name of degenerative stigmas assumed great importance in the medical psychiatric literature. Many attempts were made in that period to delimitate, classify and evaluate this group of morphologic anomalies which Régis aptly called "vices d'organisation." The enormous exaggeration of the significance of these degenerative stigmas was followed by a relative neglect of the deviations of morphologic habitus until the modern development of endocrinology. The dysfunctions of endocrine glands were found to be associated with more or less distinct morphologic anomalies, and this fact led to a renewed interest in the problems of human morphology in general. The same group of morphologic anomalies, though its confines have considerably changed, was included by Kretschmer [1] in his system of morphologic types under the designation dysplastic. Whereas the stigmas of degeneration were regarded as signs of abnormality distinguishing psychiatric patients (and "born criminals") from the normal, Kretschmer, "striving to bring the two spheres of the biologically healthy and the pathological together," understands as "dysplastic" "such forms of growth as vary very markedly from the average and commonest form of the type in question."

That these morphologic anomalies occur in different psychopathologic conditions in varying degrees has been observed in a general way for some time. Their frequency in cases of mental deficiency and epilepsy has often been pointed out. Rehm [2] and Wuth [3] remarked on their infrequency in manic-depressive psychoses. Bleuler [4] found fewer

* Submitted for publication, Oct. 25, 1928.

1. Kretschmer, E.: Physique and Character, New York, Harcourt, Brace & Company, 1925.

2. Rehm, O.: Das manisch-melancholische Irresein, Berlin, Julius Springer, 1919.

3. Wuth, O.: Konstitution und endokrines System, München. med. Wchnschr. **69**:392 (March) 1922.

4. Bleuler, E.: Affektivität, Suggestibilität, Paranoia, Halle, Carl Marhold, 1926.

"signs of degeneration" in paranoia than in schizophrenia—an opinion with which Lange [5] has disagreed—and again fewer signs in schizophrenia of paranoid type than in the other forms of schizophrenia. White,[6] on the other hand, mentioned "stigmata of degeneration" as showing sometimes the hereditary taint in paranoia and paranoid states. The frequency of dysplastic signs in schizophrenic conditions has been especially noted. The relative incidence of these dysplastic traits in different types of psychopathologic conditions, however, is not as yet well established.[7] A systematic study of this character based on a large number of uniformly diagnosed and unselected psychiatric cases, therefore, seemed desirable.

In 1896, Adolf Meyer [8] although condemning exaggerated inferences drawn from "stigmata of degeneration" advised objective and impartial observation and collection of these deviations. Interest in anthropologic habitus was continued in physical examinations at the Phipps Clinic independent of the now superseded formulations of degeneration and the one-sided endocrinologic interpretations. It was possible, therefore, to collect as the material of this study a group of 923 cases of mental disease observed in the Henry Phipps Psychiatric Clinic, in which there were recorded the presence or absence of deviations of anthropologic make-up in the sense of dysplastic signs which I propose to speak of as "growth disorders." In this group of psychiatric cases special attention had been given to the collection and evaluation of catamnestic psychiatric data, entirely independent of the purpose of this present study.

The purpose of the investigation was to determine what type of growth disorders occurs in a morphologically and psychiatrically unselected group of 923 mental patients and how these growth disorders are distributed in the different diagnostic groups.

In the grouping of the various diagnostic units two principles were especially kept in mind which are of importance in constitutional studies in psychiatry. First, a splitting up into too small groups had to be avoided as this would make statistical conclusions too fallible. The second principle concerns the two large groups of psychoses, namely, the affective (manic-depressive) and the schizophrenic, which constitute the majority of patients observed in psychiatric clinics and in which etiologically a constitutional factor is of undoubted importance. It

5. Lange, J.: Die Paranoiafrage, Leipzig, 1927.

6. White, W. A.: Outlines of Psychiatry, Washington, Nervous and Mental Disease Publishing Company.

7. The literature on the subject is summarized in the latest German edition of Kretschmer's book, "Physique and Character."

8. Meyer, Adolf: A Review of Signs of Degeneration and of Methods of Registration, Am. J. Insan. **52**:344, 1895-1896.

seemed advisable to separate those cases which formally belong to these groups from the cases which, although they might be subsumed under the diagnostic headings of schizophrenia or affective psychosis, show heterogenous clinical features. In this way, for example, psychoneurotic depressions or agitated depressions were separated from the clearcut affective psychoses, although they are clinically and constitutionally, though perhaps not formally, closely allied to the affective group.

The material therefore consists of 923 psychiatric patients distributed in the following psychosis groups:

Schizophrenic reaction type	231
Schizophrenic reaction type with affective features	27
Affective (manic-depressive) reaction type; depressions	41
Affective (manic-depressive) reaction type; manic excitements	16
Psychoneurotic depressions (including psychasthenic and reactive depressions)	36
Agitated depressions	57
Depressive conditions with various clinical admixtures	55
Paranoid psychoses	24
Paranoia	1
Affective reaction type with schizophrenic features (including excitements and depressions)	24
Organic reaction type	65
Dementia paralytica	57
Delirious reaction type (exogenic reaction type)	28
Epilepsy	108
Psychoneuroses (hysteria, neurasthenia, chronic invalidism, anxiety neuroses)	58
Psychasthenia	48
Constitutional psychopathic personality	43
Unclassified	4
	923

It is necessary to keep in mind that the delimitation of these growth disorders is a relative one. There are degrees in asymmetries and other variations of formation. Isolated minor degenerative signs such as small variations in the form of the ear or isolated minor endocrine signs like variations of the eyebrows seem to be of little significance. The growth disorders referred to in the cases of this study are uniform as only the more marked deviations were recorded. This relativity of judgment as to what in the individual case should be considered as a growth disorder affects greatly the validity of statistics as to the absolute incidence of growth disorders in mental disease. The emphasis of the present study is on the relative incidence of growth disorders in different disease groups compared with each other. In the relatively large number of cases a uniform treatment of the question as to what should be included as a growth disorder gave the possibility of determining this relative incidence a little more accurately than before. It should be emphasized, however, that even so the percentages can be considered only as approximations.

Congenital malformations of all kinds were included.[9] Care was taken to exclude bodily anomalies due to external injuries. In this connection the possibility of automutilation deserves mention. I have seen cases in which endocrinologic interpretations of scanty eyebrows were upset by the patient's admission that she had "plucked" her eyebrows a little excessively for cosmetic reasons. In one case of this material a male patient had a peculiar hair limit bordering his forehead. The hair line was even and it seemed at first as if the patient had an anomaly of hair distribution or premature baldness of an unusual character. It was then learned that he had pulled out a great deal of his hair owing to his depressive agitation.

There seems to be a gap in medical anthropologic literature concerning the question of what minor deviations of the normal adult type occur during the evolution and involution of the individual person. The inadequacy of the knowledge concerning the constitutionally important topic of hair growth and its variations may serve as an example.[10] In this study hypertrichoses of minor degree, such as hypertrichosis of "negroid type" occurring in women of advanced age, were not considered as dysplastic signs.[11] It is necessary to emphasize the importance of the age factor for the delimitation and interpretation of dysplastic signs. The delimitation of constitutional growth disorders is also not so well defined as to the adjacent field of physiologic disturbances. Amenorrhea and dysmenorrhea, frequently associated with hypoplastic pelvic genital organs, were included as growth disorders only when hypoplastic pelvic organs were found on gynecologic examination or when combined with other dysplastic traits. According to this material, severe disturbances of menstruation, especially in early life, seem to occur more frequently in schizophrenic conditions than in others. Figures as to the absolute incidence of growth disorders in the sexual sphere have to be considered with great caution, because gynecologic examination of mental patients evidently cannot be made easily as a routine. That hypoplastic genital organs are frequent in schizophrenic women has been demonstrated by Hauck.[12] Another physiologic disturbance of development is premature menopause, which also has been included here only if associated with morphologic growth disorders.

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9. A good survey of the subject of congenital malformations is found in Dubreuil-Chambardel, L.: Les variations du corps humain, Paris, Ernest Flammarion, 1925.

10. Developmental changes in the puberty period have been investigated by Scheidt. (Somatoskopische und somatometrische Untersuchungen an Knaben des Pubescenzalters, Ztschr. f. Kinderh. **28**:71, 1923.)

11. The subject of hypertrichosis has been well discussed by Danforth (Hair, with Special Reference to Hypertrichosis, Chicago, American Medical Association, 1925).

12. Hauck, quoted by Kretschmer (footnote 1).

It was considered desirable for practical reasons to mention the dysplastic signs singly, although their significance both for morphologic-diagnostic purposes and for reasons of their physiologic interpretation lies in their occurrence in combination with other signs.[13] Certain minor signs are therefore listed which if occurring alone would not be sufficient justification for including the patients among those with growth disorders. They are included because they were found frequently associated with other growth disorders. In a number of instances it was nevertheless unavoidable to mention combinations of several morphologic traits. This procedure in reality represents a middle path which seems to be justified, as it were, in its historical setting. In the period of interest in stigmas of degeneration, isolated and unrelated morphologic signs were given undue significance. In the one-sided endocrinologically oriented morphology the focus of attention was on the combination of signs, often with an oversystematization into speculative combinations and clinical syndromes.

The following growth disorders were found:

1. Skeletal anomalies:
   - Extreme height (6 feet 3 inches [190.5 cm.] in a woman)
   - General underdevelopment (both sexes)
   - Arms and legs very short in comparison with trunk length
   - Arms and legs very long in comparison with trunk length
   - General masculine build in women
   - General feminine build (wide pelvis, etc.) in men
   - Marked scoliosis
   - Marked kyphosis
   - Acromegaloid signs (prognathism, large frontal bones, etc.)
   - Deformation of skull (depressed front of skull, etc.)
   - Macrocephaly (both sexes)
   - Closed-in sella turcica (as indicated by x-rays)
   - High narrow palate
   - Wide spacing of teeth
   - Markedly tapering fingers
   - Blunt, spadelike extremities
   - Very short thick stubby fingers
   - Bilateral flat feet.
2. Asymmetries:
   - Marked asymmetries of face or chest in both sexes
3. Disproportions:
   - Eunuchoid proportions
   - Disproportionately large hands and feet
   - Short arms associated with very long hands and long legs

13. Compare discussion of dysplastic types in: Wertheimer, F. I., and Hesketh, F. E.: The Significance of the Physical Constitution in Mental Disease, Baltimore, Williams & Wilkins Company, 1926; A Minimum Scheme for the Study of the Morphologic Constitution in Psychiatry, Arch. Neurol. & Psychiat. **17**:93 (Jan.) 1927; Observations and Remarks on the Physical Constitution of Female Psychiatric Patients, Am. J. Psychiat. **6**:499, 1927.

Short legs suggestive of chondrodystrophy, occurring in a female patient with masculine type of crines
Little finger very short (reaching scarcely to middle of next finger)

4. Malformations:
   Tower skull
   Harelip, combined with pointed chin, small ears, very narrow deep palate and much thickened alveolar processes
   Markedly receding forehead with sharp-angled profile
   Mongoloid type of tongue
   Congenital malformation of left hand: flexed ring finger and rudimentary little finger
   Distal joints of second finger of left hand and third finger of right short and claw-shaped
   "Fifth finger" at second joint of little fingers of both hands
   Deformed little finger of both hands (observed in two previous generations)
   Second and third phalanges missing from right index finger; terminal phalanges of other fingers (both hands) broadened and thickened
   Hypospadias
   Spina bifida occulta
   Webbed toes, both feet
   Congenital deformity of both feet
   Fourth toe (both feet) deformed (combined with stubbing of thumbs)
   Second and third toes (both feet) paired though without a definite web
   Webbing of second and third toes of each foot
5. Anomalies of development of primary and secondary sex characteristics:
   Precocious development of genitalia (in children)
   Retarded puberty (in children)
   Undersized penis or testes (or both)
   Atrophic testes
   One testis (or both) undescended
   Delayed development of secondary sex characteristics (both sexes)
   Pronounced feminine type of fat distribution and body hair distribution in men
   Amenorrhea (for example, patient of 49 who had never menstruated)
   Markedly delayed menarche
   Premature menopause
   Vicarious menstruation associated with late sexual maturity
   Prominent mammae and mons Veneris in men
   Underdeveloped or atrophied breasts in women
   Hypertrophy of breasts and labia majora (combined with atrophic clitoris and masculine crines)
   Hypertrophic labia minora with atrophic breasts and masculine crines
   Infantile uterus with hypoplastic genitalia
   Hypoplasia of uterus
   Underdeveloped external genitalia in women (with or without scanty labial hair)
   Infantile uterus with delayed puberty
   Poorly developed secondary sex characteristics in both sexes
6. Anomalies of glands other than sex glands:
   Enlarged thyroid considered only when associated with other morphologic signs

7. Anomalies of fat distribution:
   - Extreme obesity
   - General obesity (in men or women) associated with endocrinologic signs
   - Obesity of feminine type in men
   - Suggested habitus lipodystrophicus
8. Anomalies of hair distribution:
   - General hypertrichosis in men or women
   - General hypotrichosis in men and women
   - Heavy nasal brows
   - Hypotrichosis of face in men
   - Hair growing very low on forehead and temples
   - Circumscribed hypotrichosis in men and women (crines, axillary hair, etc.)
   - Complete baldness at 29 years in woman without eyebrows or eyelashes
   - Circumscribed hypertrichosis in women (legs and arms, face and chin, etc.)
   - Premature gray hair in women (at ages of 12, 16, 32, 33)
   - Hair distribution of masculine type in women
   - Hair distribution of feminine type in men
9. Anomalies of complexion (skin):
   - Harsh rough skin, ichthyosis
   - Myxedematous type of skin
   - Overpigmentation in circumscribed areas (around areolae, axillary borders, around eyes, etc.)

The following cases are given briefly to illustrate typical cases with growth disorders.

REPORT OF CASES

CASE 1.—A woman, aged 22, of the schizophrenic reaction type, was 5 feet 2½ inches (158.6 cm.) tall and weighed 92 pounds (41.7 Kg.). The thyroid was palpable and the pulse rate was 120. She showed some growth of coarse hair on the chest and about the areolae, heavy growth in the axillae and on the legs and a distinct downy growth on the chin and upper lip. The pubic hair showed masculine distribution with the line to the umbilicus well marked. Her trunk was slim, firm and boyish, with narrow hips and undeveloped breasts.

CASE 2.—A man, aged 31, of the schizophrenic reaction type, was 5 feet 11 inches (180.3 cm.) tall and weighed 178 pounds (80.7 Kg.). His body was of the female type, with narrow shoulders, prominent breasts and very fat hips, thighs and abdomen. The distribution of pubic hair approached the female type. The genitalia were small and undeveloped. Roentgen examination showed a closed-in sella turcica.

CASE 3.—A woman, aged 31, with agitated depression, presented a marked overgrowth of hair on the face and chin; the eyebrows were heavy and met in the midline; there was hypertrichosis of the trunk, especially the shoulders. The distribution of the pubic hair was of the masculine type. The thyroid isthmus was palpable.

CASE 4.—A woman, aged 40, with paranoid schizophrenic psychosis, was short and stocky, of strong build. Her skin was pale, coarse and thick; she had a stubby nose and rather short and dull features. Her extremities were blunt and spadelike. Her general appearance suggested a hypothyroid condition.

CASE 5.—A woman, aged 30, of the schizophrenic reaction type, was 4 feet 9 inches (144.8 cm.) tall and weighed 56 pounds (25.4 Kg.). She was generally emaciated. Her lower jaw was weak and receding. Her hair grew low on the

forehead and was thin and fine. Her eyebrows were very scanty. Her skin was of a waxy whiteness and showed the underlying network of veins. The mammary glands were undeveloped. The pubic and axillary hair was scanty. The external genitalia were undeveloped, and the pelvic organs were infantile.

Enlargement of the thyroid was considered only as a morphologic sign, especially in association with other morphologic signs, and not from the physiologic point of view. Statements with regard to slight changes of size of the thyroid have to be evaluated with great caution. The incidence of the milder thyroid disorders is not without importance, but it can be attacked only from the clinical physiologic side. There are marked differences of opinion among authoritative clinicians with regard to the palpation of the thyroid, as has been recently pointed out again by Wagenseil [14] in an important paper on eunuchism and eunuchoidism; according to some (i.e., Liek [15]) the normal thyroid can be neither seen nor felt; according to others (i.e., de Quervain [16]) it can be clearly palpated, if the fat and musculature of the neck are not unusually strongly developed.

The bulk of these growth disorders constitutes a heterogeneous group. One might attempt to distinguish among them tentatively three broad groups: (1) definite congenital malformations; (2) growth disorders which constitute the more typical endocrinologic syndromes, either full-fledged or "mitigated" (Oswald [17]); (3) growth disorders with similarity to endocrinologic disorders but occurring in clinically not well definable types. For the last two groups, however, this division could be carried out only with clinical and physiologic methods and not by morphologic criteria alone.

The distribution of growth disorders in the different diagnostic groups is given in tables 1 and 2.

Of the total number of 923 mental patients, 194, or 21 per cent, showed growth disorders. The largest incidence of growth disorders is clearly in the group of schizophrenic psychoses [18] (36.4 per cent). It

14. Wagenseil, F.: Beiträge zur Kenntnis der Kastrationsfolgen und des Eunuchoidismus beim Mann, Ztschr. f. Morphol. u. Anthrop. **26**:264, 1927.

15. Liek, quoted by Wagenseil (footnote 14).

16. de Quervain, quoted by Wagenseil (footnote 14).

17. Oswald, A.: Aus der klinischen Pathologie der innerren Sekretion: Die mitigierten Formen der klassischen Syndrome, Schweiz. med. Wchnschr. **56**:993, 1926.

18. This figure is more significant if it is taken into consideration that in this study only the more marked growth disorders as found in the medical examinations of the patients were included. There is reason to believe that when the less conspicuous dysplastic signs are taken into account the incidence of growth disorders in schizophrenic psychoses is even greater.

has to be remarked that the number of schizophrenic cases (231) is greater than that of any other group. The next largest incidence of growth disorders occurs in the paranoid psychoses, 29 per cent of

TABLE 1.—*Distribution of Growth Disorders Among 923 Cases*

1. According to the Diagnostic Groups

Diagnosis Group	Total Number of Cases	Percentage of All Cases	Patients With Growth Disorders	Patients Without Growth Disorders
Schizophrenic reaction type	231	25.0	84	147
Schizophrenic reaction type with affective features	27	2.9	7	20
Affective (manic-depressive) reaction type; depressions	41	4.5	1	40
Affective (manic-depressive) reaction type; manic excitements	16	1.8	0	16
Psychoneurotic depressions (including psychasthenic and reactive depressions)	36	3.9	4	32
Agitated depressions	57	6.2	12	45
Depressive conditions with admixtures	55	6	7	48
Paranoid psychoses	24	2.6	7	17
Paranoia	1	0.1	0	1
Affective reaction type with schizophrenic features (excitements and depressions)	24	2.6	5	19
Organic reaction type	65	7	4	61
Dementia paralytica	57	6.1	5	52
Delirious reaction type (exogenic reaction type)	28	3	4	24
Epilepsy	108	11.7	28	80
Psychoneuroses	58	6.3	8	50
Psychasthenia	48	5.2	7	41
Constitutional psychopathic personality	43	4.7	9	34
Unclassified	4	0.4	2	2
	923		194	729

TABLE 2.—*Distribution of Growth Disorders Among 923 Cases*

2. In Each Diagnostic Group

Diagnosis Group	Patients With Growth Disorders		Patients Without Disorders	
	Number	Percentage	Number	Percentage
Schizophrenic reaction type	84	36.4	147	63.6
Schizophrenic reaction type with affective features	7	26.0	20	74.0
Affective (manic-depressive) reaction type; depressions	1	2.4	40	97.6
Affective (manic-depressive) reaction type; manic excitements	0	0	16	100.0
Psychoneurotic depressions (including psychasthenic and reactive depressions)	4	11.1	32	88.9
Agitated depressions	12	21.0	45	79.0
Depressive conditions with admixtures	7	12.7	48	87.3
Paranoid psychoses	7	29.0	17	71.0
Paranoia	0	0	1	100.0
Affective reaction type with schizophrenic features (excitements and depressions)	5	20.8	19	79.2
Organic reaction type	4	6.0	61	94.0
Dementia paralytica	5	9.0	52	91.0
Delirious reaction type (exogenic reaction type)	4	14.3	24	85.7
Epilepsy	28	26.0	80	74.0
Psychoneuroses	8	13.8	50	86.2
Psychasthenia	7	14.6	41	85.4
Constitutional psychopathic personality	9	21.0	34	79.0
Unclassified	2		2	

twenty-four cases. There follow epilepsy, 26 per cent of 108 cases; schizophrenic psychoses with affective features, 26 per cent of twenty-seven cases, and agitated depressions, 21 per cent of fifty-seven cases.

In the paranoid group cases were also included which are closely allied to or might be considered to belong to the schizophrenic group.

The lowest incidence of growth disorders is in the group of manic excitements, with no disorders occurring in sixteen cases. The next group is that of depressions, with only one of forty-one patients having growth disorders. If the percentage is calculated for the affective reaction type including both manic excitements and depressions, it is found that there is only one of fifty-seven patients, or 1.8 per cent, with growth disorders. Then follow organic reaction types, 6 per cent of sixty-five cases, and the separately counted group of dementia paralytica, which belongs to the organic reaction type, with 9 per cent of fifty-seven cases. As to paranoia, a clinically rare condition according to strict clinical standards, the one case occurring in the material was free from growth disorders. It is then possible to say that, according to the material, schizophrenic and epileptic patients show most growth disorders, depressions and manic excitements least.

TABLE 3.—*Ratio Between the Manic-Depressive and the Schizophrenic Groups*

Diagnosis	Growth Disorders	No Growth Disorders	Total	Percentage of Growth Disorders in Total
Affective (manic-depressive).....	1	56	57	1.75
Schizophrenic.....	84	147	231	36.4

Of particular interest is the relationship between schizophrenic psychoses and manic-depressive psychoses, the two large functional constitutional psychotic groups. As in any unselected material based on patients observed in a psychiatric clinic, the number of schizophrenic psychoses is greater than the number included in the manic-depressive group. If the pure cases and those most clearly corresponding to the types of these two great reaction forms are selected, the ratio between them is that indicated in table 3.

The difference in the occurrence of growth disorders indicated in table 3 cannot be explained by chance and points definitely to a biologic correlation. The chi square (χ^2) for this fourfold table (computed by Dr. H. L. Dunn) is 26, demonstrating that the probability of obtaining a sampling difference of 34.6 per cent in growth disorders between manic-depressive and schizophrenic psychoses is only four chances in a million.

If one includes in the two groups of affective and schizophrenic psychoses those cases which correspond less closely with the formal clinical types and which in the final diagnosis of the clinic have been subsumed under separate headings, the percentages, especially for the affective reaction type, are somewhat different.

In table 4, with the schizophrenic psychoses are included the paranoid psychoses and schizophrenic psychoses with affective features. In the affective group are included, beside depressions and manic excitements, depressions with other clinical admixtures, agitated depressions and depressions and excitements wtih schizophrenic features.

If tables 3 and 4 are compared, it is seen that the percentage of growth disorders in the schizophrenic group remains almost the same when conditions allied to schizophrenia are added. In the manic-depressive group, on the other hand, the percentage changes considerably when the less clear affective psychoses are added. This change in the percentage of the larger group of affective psychoses is due mainly to three groups: (1) affective reaction type with schizophrenic features; five patients with growth disorders, nineteen patients without growth disorders; (2) depressions with clinical admixtures; seven patients with growth disorders, forty-eight pateints without growth disorders; (3)

TABLE 4.—*Ratio Between Manic-Depressive and Schizophrenic Groups with Allied Conditions*

Diagnosis	Growth Disorders	No Growth Disorders	Total	Percentage of Growth Disorders in Total
Affective reaction type and allied conditions	29	200	229	12.6
Schizophrenic reaction type and allied conditions	98	184	282	34.8

agitated depressions; twelve patients with growth disorders, forty-five patients without growth disorders.

The difference between clear depressions of manic-depressive stamp and agitated depressions, as regards the incidence of growth disorders, seems especially noteworthy. These two conditions occur in this material in well comparable numbers: depressions, forty-one; agitated depressions, fifty-seven. The frequency of growth disorders in the agitated depression group is about ten times greater than in the group of simple depressions. It would seem that the distinction between simple depressions and agitated depressions which has been worked out from the psychiatric symptomatologic point of view may have also a wider biologic bearing.

In a separate calculation of those growth disorders falling under the group of congenital malformations, it was found that this smaller group is scattered through all the psychiatric diagnosis groups and follows the distribution occurring in the whole group of growth disorders. The maximum incidence of congenital malformations is in the schizophrenic group. The fact that the incidence of congenital malformations conforms on the whole with the distribution of the dysplastic signs (in the

narrower sense) is of considerable interest. It justifies the treatment of morphologic deviations as a whole under the inclusive designation of "growth disorders," as has been suggested in this study.

The contrast in the incidence of bodily growth disorders between schizophrenic and affective patients has a striking parallelism in the sphere of mental development. Just as patients with manic-depressive psychoses show a tendency toward unstunted and harmonious bodily growth, as is clearly evident from this study, so they are also usually personalities with likewise harmonious mental maturation. The affective psychosis interrupts this mental development, but in most cases does not essentially deflect it. Schizophrenic patients, on the other hand, who show the largest number of disorders of physical growth, frequently show also in their prepsychotic mental development traits of inharmonious growth, immaturity and combinations of mental precocity and arrested development in intellectual, emotional and instinctive life. The psychosis frequently affects deeply and blocks the further growth of the personality. Both the type of the psychosis and the physical growth disorders are the expression of a certain constitutional biologic make-up.

SUMMARY

1. Under the term "growth disorders" are included the more marked abnormalities of bodily configuration, which have been variously described as stigmas of degeneration, dysplastic signs, errors of development, etc.

2. In 923 unselected psychiatric patients a series of growth disorders was found which are enumerated under the following headings: skeletal anomalies, asymmetries, disproportions, malformations, anomalies of development of primary and secondary sex characteristics, anomalies of glands other than sex glands, anomalies of fat distribution, anomalies of hair distribution and anomalies of complexion (skin).

3. Among these growth disorders three broad groups can be tentatively distinguished: (1) definite congenital malformations; (2) growth disorders which constitute the more typical endocrinologic syndromes, either full-fledged or "mitigated" (Oswald); (3) growth disorders with similarity to endocrinologic disorders but occurring in clinically not well definable types. For the last two groups, however, this division could be carried out only with clinical and physiologic methods and not by morphologic criteria alone.

4. Of 923 patients, 194, or 21 per cent, showed growth disorders. The largest incidence was in schizophrenic psychoses (36.4 per cent), followed by paranoid psychoses (29 per cent), epilepsy (26 per cent) and agitated depressions (21 per cent). The lowest incidence was in manic excitements (no case of sixteen), followed by depressions (one

case of forty-one) and organic reaction type (6 per cent). The one case of paranoia in the material was found to be without growth disorders.

5. The proportion in the incidence of growth disorders between the clear cases of affective (manic-depressive) and schizophrenic psychoses is 1.7 per cent to 36.4 per cent. Statistical computation shows that this difference of 34.6 per cent cannot be explained as due to chance sampling and that it therefore points definitely to a biologic correlation.

6. Ten times as many cases with growth disorders occur in agitated depressions as in simple depressions of manic-depressive type.

7. The distribution of congenital malformations among the various psychopathologic groups conforms generally with that of the whole group of growth disorders.

8. There is a certain parallelism between the frequency and rarity of bodily growth disorders in schizophrenic and affective psychoses, and the occurrence and absence of disharmonies in the mental development of the prepsychotic personalities suffering from these conditions.

DANGERS OF DIAGNOSTIC LUMBAR PUNCTURE IN INCREASED INTRACRANIAL PRESSURE DUE TO TUMOR OF THE BRAIN

A REVIEW OF TWO HUNDRED CASES IN WHICH LUMBAR PUNCTURE WAS DONE*

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The belief is widespread among neurologic surgeons and others that lumbar puncture in patients with increased intracranial pressure due to tumor of the brain is a dangerous and unwarranted procedure. At the New York Neurological Institute there has never been any hesitation at performing lumbar puncture for diagnostic purposes, unless the signs and symptoms pointed to a subtentorial new growth. It seemed, therefore, that it would be of interest to review 200 of the cases in which this procedure was followed.

The literature on the danger of lumbar puncture in states of increased intracranial pressure is not large, and I was able to find but few studies of large series of cases. In most instances, I found only isolated reports of single cases, in which untoward symptoms and even death occurred after this procedure. Numerous writers spoke of the dangers of lumbar puncture in the presence of tumor of the brain, without giving any details.

Cushing,[1] in a paper on "Some Aspects of the Pathological Physiology of Intracranial Tumors," made the following statements:

> In all cases in which pressure phenomena are present and the obstructed fluid has acquired an increased degree of tension, it collects and distends the basilar cysternae, finding its way into the optic sheaths; hence the choked disc. It also passes into the olfactory nerves; hence its occasional escape from the nares (rhinorrhea); and into the spinal canal where it may be demonstrated under increased tension if one wishes to take the risk of performing a lumbar puncture in those cases.
>
> In conclusion, one recognized characteristic of the brain under pressure is its tendency to herniate through a cranial defect, and as there is normally an opening at the foramen magnum, a certain degree of protrusion is usually present there. In the presence of such conditions the withdrawal of the cerebrospinal fluid from the spinal meninges by a lumbar puncture is often hazardous, as it may tend to a sudden wedging of the bulb in the opening, with anemia and paralysis of the vital centers.

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* From the Neurosurgical Clinic (Dr. Elsberg) and the Neurological Divisions of the New York Neurological Institute.

1. Cushing, H.: Some Aspects of the Pathological Physiology of Intracranial Tumors, Boston M. & S. J. **161**:71 (July 15) 1909.

Comfort,[2] in a report of seventy-three cases of tumor of the brain, thirty-five of which were verified at operation or at autopsy, and in all of which lumbar puncture had been performed, did not mention any instance of dangerous symptoms or of death following this procedure.

Conner [3] stated that "in a few cases of uremia and brain tumor death has followed within a few hours the withdrawal of a large amount of fluid." "Those conditions in which the greatly increased cerebrospinal fluid pressure has existed for some time, e. g., brain tumors—are the ones apparently which bear least well the sudden removal of a large quantity of fluid." This is a warning against a medical decompression, but should not be interpreted as prima facie evidence against a diagnostic puncture, even in a case of tumor of the brain in which long continued increased intracranial pressure has existed.

Frazier [4] spoke of the use of lumbar puncture, during cranial operations, to relieve intracranial pressure. In one case, 50 cc. of spinal fluid was removed to enable the operator to open the dura.

TABLE 1.—*Material Studied with a View to Ascertain the Danger of Lumbar Puncture in the Presence of Tumor of the Brain*

Tumor Suspected Present	Total Number of Cases	Cases Verified	Cases Not Verified
Supratentorial new growths	141	..	..
Papilledema not present	39	19	20
Papilledema present	102	43	59
Infratentorial new growths	59	..	..
Papilledema not present	17	8	9
Papilledema present	42	24	18

MATERIAL

The material on which this report is based is set forth in table 1. The 200 cases mentioned in table 1 are not a selected group. Two hundred consecutive patients with increased intracranial pressure due to proved or presumed tumor of the brain, in whom lumbar puncture was performed, were observed as the basis for the study. In the majority of the cases of tumor verified by operation or by autopsy, the growth was a glioma of one type or another, although neurinomas, meningeal fibroblastomas (meningiomas, endotheliomas) and other types of intracranial neoplasms are represented in the series. The growths were located in various parts of the brain. Cases of subtentorial tumors were included, although the question of the danger of lumbar puncture in

2. Comfort, M. W.: Yellow Spinal Fluid Associated with Tumor of the Brain, Arch. Neurol. & Psychiat. **15**:751 (June) 1926.

3. Conner, L. A.: The Technique of Lumbar Puncture, New York M. J. **71**:723 (May) 1900.

4. Frazier, C. H.: The Cerebrospinal Fluid and Its Relation to Brain Tumors, New York M. J. **99**:1275 (June 27) 1914.

the presence of a tumor in the posterior cranial fossa is quite another matter, and lumbar puncture in subtentorial expanding lesions is probably always contraindicated, except after operations in which a suboccipital craniotomy with removal of the margin of the foramen magnum and a wide incision of the dura has been performed.

In what follows, an effort has been made to correlate the nature of the neoplasm, its location and size, the roentgen evidence of increased intracranial pressure, the degree of papilledema, if present, the intraventricular pressure of the cerebrospinal fluid (when measured), the tension of the dura in the patients subjected to operation, the spinal manometric pressure (when measured) and, finally, the amount of fluid removed by lumbar puncture (whenever this was definitely recorded), so as to arrive at a conclusion regarding the actual danger of lumbar puncture, if there is any danger, in states of increased intracranial pressure due to suspected tumor of the brain.

In the majority of the 200 cases, not more than 5 cc. of cerebrospinal fluid for examination was withdrawn by puncture of the spine, and when the fluid was found escaping under marked increase of pressure the stylet was reintroduced into the needle, and the fluid was allowed to drop out slowly. The needles used for the puncture were of fine caliber and of the Quincke type. It is hardly necessary to mention that needles of coarse caliber should never be used. A needle of large caliber might allow fluid, under greatly increased pressure, to escape so quickly as to bring about a sudden marked change in intracranial pressure, such that serious symptoms might be caused and even a fatality from the sudden diminution of pressure within the skull. Lumbar puncture in the cases reviewed was limited to a diagnostic procedure done with the patient lying in the right or the left lateral prone position, and with the patient kept in the supine position for at least twenty-four hours afterward. In some instances, however, confused patients have, despite great care, got out of bed soon after the puncture, without, however, untoward results.

OBSERVATIONS

Lumbar Puncture in Verified Tumors.—1. Infratentorial Expanding Lesions (32 Cases Verified by Operation or by Autopsy): In patients with infratentorial expanding lesions, the puncture was performed before the diagnosis of a growth in the posterior fossa had been made and proved by operation or by autopsy. The results of lumbar puncture in these patients are recorded in table 2. The results show that not even in subtentorial expanding lesions were any serious untoward effects produced by lumbar puncture and the removal of a small amount of cerebrospinal fluid. If, at the time, the diagnosis of a subtentorial expanding lesion had been made or suspected, a lumbar puncture would not and should not have been performed.

This group of cases is of great interest, showing, as it does, that even in the presence of a tumor in the posterior cranial fossa the danger of lumbar puncture is not great. Notwithstanding the results in this series, a sufficient number of cases in which serious symptoms and death followed the withdrawal of fluid by puncture of the spinal subarachnoid space have been recorded in the literature to indicate that this procedure should not be carried out if a subtentorial expanding lesion is diagnosed or suspected.

TABLE 2.—*Results of Diagnostic Lumbar Punctures in Verified Infratentorial Tumors*

Location and Character of New Growth; and the Associated Conditions	Number of Cases	Number Times Lumbar Puncture Was Performed	Results
1. Without Papilledema: 8 Cases			
X-ray negative; spinal fluid not under pressure	4	4	In 3 cases, no effect; in 1, headache but not severe
Convolutional atrophy; headache, drowsiness; evidence of pressure	4	4	In 3 cases, no change; in 1, headache but not severe
2. With Papilledema (of from +2 to +7 Diopters): 24 Cases			
At operation, tension of dura not increased; no evidence of pressure	1	1	Slight nausea day before puncture, aggravated for two days thereafter
Mucoid cyst of the arachnoid above the foramen magnum, lying on the medulla; evidence of pressure	1	5	Followed by slight headache 3 times; by none 2 times
Left cerebellar abscess with 60 cc. of pus; evidence of pressure	1	1	12 cc. of fluid removed; no new symptoms following; no headache
Meningeal fibroblastoma in lateral recess; evidence of pressure	1	1	Puncture made with patient in sitting position; 5 cc. removed; for a few minutes, patient became pale and perspired freely; pulse grew rapid; patient did not faint; spinal fluid yellow
Endotheliomas, gliomas, tumors of the nervus acousticus, etc.; evidence of pressure	20	22	In 18 cases, no effect; in 3, headache for a day or two; in 1, severe headache; in 2, two punctures were performed before operation without ensuing new symptoms

2. Supratentorial Tumors (62 Cases Verified by Operation or by Autopsy): The results of lumbar puncture in this group are recorded in table 3. One additional case deserves more extended notice.

A patient had been operated on and an irremovable spongioblastoma multiforme had been exposed in the left temporal and parietal lobes. Only a specimen had been removed for verification, and a large defect in the bone had been left for decompressive purposes. The patient had been readmitted to the hospital several times, some relief being obtained by the removal of fluid by lumbar puncture. Then the patient was admitted again and, on lumbar puncture and withdrawal of a considerable amount of fluid, the bulging cerebral hernia suddenly flattened, the pulse and the respirations became shallow and the patient remained in a condition of collapse for ten minutes, after which she recovered.

This was the most serious effect from lumbar puncture that was seen in the entire series of supratentorial expanding lesions here recorded. In this case, it must be remembered, however, that the lumbar puncture was not performed for diagnosis, but for its decompressive effect, and the temporary symptoms were the result of a therapeutic and not of a diagnostic procedure.

TABLE 3.—*Results of Diagnostic Lumbar Punctures in Sixty-Two Cases of Verified Supratentorial Tumors*

Location and Character of New Growth; and the Associated Conditions	Number of Cases	Number Times Lumbar Puncture Was Performed	Results
1. Without Papilledema: 19 Cases			
Pituitary tumors; no evidence of increased intracranial pressure	2	2	In 1 case, no effect; in 1, slight headache
Endothelioma; with other signs of increased intracranial pressure	3	3	In 2 cases, no effect; in 1, headache, nausea and vomiting for 3 days
Glioma; spinal fluid under pressure equal to 70 mm. of water	1	1	No change
Glioma; spinal fluid under pressure equal to 360 mm. of water	1	1	Headache increased for 48 hours
Glioma; dura found tense at operation	6	7	In 6 cases, no change; in 1, headache for 4 days
Fibrosarcoma, gumma, etc.	6	6	No change
2. With Papilledema: 43 Cases			
Suprasellar tumor; papilledema of +4 diopters	2	2	In 1 case, no change; in 1, headache increased
Endothelioma; papilledema to +6 diopters	11	11	In 8 cases, no change; in 3, headache for from 24 to 48 hours
Cholesteatoma; papilledema of +5 diopters	1	2	Punctures performed 2 years apart; no effect from either puncture
Glioma (spongioblastoma, astrocytoma and other ripe and unripe forms); papilledema of low grade up to +1 diopter	6	7	In 4 cases, no change; in the fifth case, in which 2 punctures were made before operation, spinal fluid was under pressure of 500 mm. of water; in the sixth case, there was a pressure of over 580 mm.; no effect in either case noted
Glioma; papilledema of from +2 to +7 diopters	20	20	In 19 cases, no change; in 1, severe headache for several days; in 1, spinal fluid not under increased pressure; in 19, pressure was above normal; in 2, as high as 520 mm. of water
Sarcoma, etc.; papilledema of from +3 to +5 diopters	3	3	In 1, no change; in 1, headache worse for 2 days; in 1, headache relieved

Summary.—In ninety-four patients with verified supratentorial and infratentorial expanding lesions within the cranial cavity, serious untoward results did not follow lumbar puncture and the removal of a small amount of cerebrospinal fluid. There was not one fatality and, with the one exception noted, the only result of the puncture was an increase of headache in nineteen patients (in most of them this was only a slight increase), nausea in two patients and faintness in one. In the one exception, the lumbar puncture and withdrawal of fluid was not done as a diagnostic but as a therapeutic measure; therefore the case does not properly belong in this discussion.

Lumbar Puncture in Unverified Tumors and Expanding Lesions.—1. Possible Infratentorial Expanding Lesion (27 Cases): The results of diagnostic lumbar puncture in these cases are summarized in table 4.

2. Suspected Supratentorial Expanding Lesions (78 Cases): The results of diagnostic puncture in these cases are set forth in table 5. In addition, there was one case that should receive special mention.

TABLE 4.—*Results of Diagnostic Lumbar Puncture in Twenty-Seven Cases of Possible Infratentorial Expanding Lesions*

Location and Character of New Growth; and the Associated Conditions	Number of Cases	Number Times Lumbar Puncture Was Performed	Results
1. Without Papilledema: 10 Cases			
Fourth ventricle (?); no evidence of pressure	1	1	No change
Cerebellum, pons varolii and nervus acusticus (?); no evidence of pressure	4	4	No change
Posterior fossa suspect; no evidence of pressure	1	1	Headache increased; vomiting for 24 hours
Cerebellum; marked hydrocephalus; no evidence of pressure	1	1	Marked increase in headache
Cerebellum and brain stem; spinal fluid under pressure	1	1	Headache made worse
Questionable glioma or tuberculoma; spinal fluid under pressure equal to 240 mm. of water	1	1	No change
2. With Papilledema Causing Slight Blurring of Disks: 3 Cases			
Tuberculoma (?); fluid under pressure	1	1	No change
Angle tumor (?); evidence of pressure	1	1	No change
Cerebellum (?); evidence of pressure	1	1	No change
3. With Papilledema of from +2 to +6 Diopters: 15 Cases			
Cerebellum; evidence of pressure	1	1	Vomiting, pain in back of neck and head
Posterior fossa; evidence of pressure	1	2	No change; one puncture with patient in sitting posture; one with patient lying down
Posterior fossa; evidence of pressure	7	7	No change
Posterior fossa; spinal fluid under considerable pressure	1	1	No change
Posterior fossa; evidence of pressure	1	1	Headache, nausea and vomiting
Posterior fossa; evidence of pressure	1	1	Slight headache
Posterior fossa; evidence of pressure	1	1	Increased headache at the time
Posterior fossa; evidence of pressure	1	1	Severe headache for 48 hours
Cerebellum and midbrain; tuberculoma (?); evidence of pressure	1	3	No change; pressure equal to 380 mm. of water

REPORT OF CASE

History.—G. M., aged 22, was admitted to the hospital on Aug. 26, 1926, complaining of weakness of the left side of the body, headache and blurred vision. Her illness began on March 4, 1925, with a sudden feeling of impending calamity, and was progressive in its course. While playing cards, she suddenly felt greatly depressed for several minutes. The next morning she had a similar attack and after that she had from two to three attacks a week.

On April 15, 1925, the patient's mother found her in bed unconscious in convulsive movements. She had seven such attacks following that date, often occur-

TABLE 5.—*Results of Diagnostic Lumbar Puncture in Seventy-Eight Cases of Suspected Supratentorial Expanding Lesions*

Location and Character of New Growth; and the Associated Conditions	Number of Cases	Number Times Lumbar Puncture Was Performed	Results
1. Without Papilledema: 20 Cases			
Dura tense; ventricular system collapsed; evidence of pressure	1	1	No change
Spinal fluid under increased pressure	2	2	No change
Right frontal lobe; evidence of pressure	1	1	No change
Right temporal lobe abscess; spinal fluid under increased pressure	1	2	No change
Pituitary (?); evidence of pressure	1	1	Headache for several days
Localization (?); no evidence of pressure	5	5	No change
Frontal lobe (?); no evidence of pressure	1	1	Headache for 3 days
Frontal lobe; no evidence of pressure	1	1	Confused as is usual after lumbar puncture
Parietal lobe (?); no evidence of pressure	1	1	No change
Frontal and parietal lobes (?); no evidence of pressure	1	1	Jacksonian attacks in right side of body continued
Tuberculoma (?); no evidence of pressure	1	1	No change
Pituitary (?); no evidence of pressure	2	2	In 1 case, no change; in 1, typical postlumbar puncture headache
Localization (?); no evidence of pressure	1	1	Slight headache for 1 day
Frontal lobe; no evidence of pressure	1	1	No change
2. With Papilledema of from +1 to +7 Diopters: 58 Cases			
Beginning papilledema; evidence of pressure	2	2	No change
Right temporosphenoidal lobe; no evidence of pressure	3	4	In 2 cases, no change; in 1 case in which 2 punctures were made, no change
Right frontal lobe; evidence of pressure	3	3	No change
Right frontal lobe; fluid in right posterior horn under pressure equal to 580 mm. of water	1	1	Puncture done with patient in Trendelenberg position; severe headache for 1 day
Right frontal lobe; dura white and tense at operation; evidence of pressure	1	1	Patient felt better
Right frontal lobe; spinal fluid under pressure equal to 380 mm. of water	1	1	Headache and vomiting for 2 days
Left frontal lobe; evidence of pressure	5	5	No change
Left frontal lobe; evidence of pressure	1	1	Headache for 3 days
x/ Left frontal lobe; papilledema of +6 diopters; evidence of pressure	1	2	In 1 case, no effect; in 1, severe headache
Bilateral frontal tumor (?); papilledema of +5 diopters; evidence of pressure	1	1	No change
Left hemisphere involved; evidence of pressure	2	2	No change
Left hemisphere involved; increased density back of left orbit; lumbar puncture showed increased pressure	1	1	No change
Left hemisphere; deep-seated lesion; markedly increased tension of dura; evidence of pressure	1	1	No change
Right frontal and parietal lobes; marked increase in tension of dura; evidence of pressure	2	2	No change
Right frontal and parietal lobes; exceedingly tense dura; lumbar puncture showed pressure equal to 280 mm. of water	1	1	No change

TABLE 5.—*Results of Diagnostic Lumbar Puncture in Seventy-Eight Cases of Suspected Supratentorial Expanding Lesions—Continued*

Location and Character of New Growth; and the Associated Conditions	Number of Cases	Number Times Lumbar Puncture Was Performed	Results
2. With Papilledema of from +1 to +7 Diopters: 58 Cases—Continued			
Left parietal lobe; spinal fluid under pressure equal to 220 mm. of water	1	1	Puncture performed with patient in sitting position; no change
Right frontal and parietal lobes; papilledema of +2 diopters; evidence of pressure	1	1	20 cc. of fluid removed each time; improvement
Right frontal and parietal lobes; evidence of pressure	1	1	Vomiting, headache and irrational actions, as before lumbar puncture
Right frontal and parietal lobes; evidence of pressure	1	1	No change
Right parietal and temporal lobes; evidence of pressure	1	1	No change
Left parietal lobe; deep infiltrating tumor (?); at operation, dura found very tense; evidence of pressure	1	1	Headache the same as before lumbar puncture
Pituitary (?) (blind); evidence of pressure	1	1	No change
Brain tumor (?) unlocalized; evidence of pressure	5	5	No change
Skull bones atrophied; evidence of pressure	1	1	No change
Sella almost completely destroyed; evidence of pressure	1	1	No change
Lumbar puncture showed pressure equal to 420 mm.; right and left posterior horns, small amount of fluid	1	1	No change
Not localized; evidence of pressure	1	1	Headache for 3 days
Not localized; brain moderately tense; papilledema of +5 diopters; signs of pressure	1	1	No change
Not localized; lumbar puncture showed "tremendous pressure"	1	1	No change
Not localized; spinal fluid under pressure equal to 160 mm. of water	1	1	No change
Not localized; patient aged 2½ years; marked signs of pressure	1	1	No change
Not localized; papilledema of +4 diopters; signs of pressure; convolutional atrophy	1	1	No change
Not localized; patient almost blind; evidence of pressure	1	1	No change
Pituitary; papilledema; no other evidence of pressure	1	1	No change
Right parietal lobe; papilledema of +5 diopters; no signs of increased intracranial pressure at operation	1	1	No change
Left parietal lobe; early papilledema; no others signs of pressure	3	3	No change
Frontal lobe (abscess); papilledema; no other evidence of pressure	1	4	No change
Right frontal lobe; spinal fluid not under tension; dura not under tension; papilledema of +6 diopters	1	1	No change in blood pressure, pulse rate or respirations
Right frontal lobe; papilledema of +4 diopters; no other signs of pressure	1	1	Severe headache and nausea for 3 days
Right frontal lobe; no signs of pressure at operation; papilledema of +3 diopters	1	1	No change
Third ventricle tumor; papilledema; no other evidence of pressure	1	1	No change

ring with her menstrual periods. In September, 1925, after swimming, she had a sudden severe headache and could not walk, having to be carried into a house. It was found that both sides of the body were weak. The power on the right side soon returned, but the left side remained weak. In August, 1926, she had another attack, followed by weakness of the left side of the face and the left arm and leg, and after that she often had headaches with attacks of projectile vomiting. The weakness of the left side grew more marked. She later complained of photophobia and poor vision.

Neurologic Examination.—Although the patient had been in bed for three weeks previous to her admission to the hospital, she was able to walk to her room with assistance. She was able to stand on each foot alone, but could use the right foot and leg better than the left. There was marked weakness of the left hand grip and diminished power of extension and flexion of the left foot. The finger-to-nose test was done normally on the right but with a slight wavering on the left; the finger-to-thumb test was slowly performed on the left, but normally carried out on the right; the heel-to-knee and along shin test was normally performed on the right, and much more slowly carried out on the left. Distinct ataxia was not observed. Some diminution of the check element in the left arm was noted with some adiadokokinesis on the left. Speech was normal. The deep reflexes were slightly more active on the left side of the body; the abdominal reflexes were less active on the left. There were plantar flexion of the right foot and a questionable Babinski sign on the left. Sensation was normal in all modalities. Cranial Nerves: A questionable quadrantic defect was present in the left upper fields of both eyes; marked papilledema was observed, greater on the left than on the right. The pupils were 3 mm. in size and reacted well to light and in accommodation; convergence was normal. There was weakness of the right sixth nerve, and a few nystagmoid jerks were observed on extreme lateral gaze. The left corneal reflex was slightly diminished. There was distinct weakness of the left lower part of the face, both voluntary and emotional. The other cranial nerves did not show any abnormality.

Course.—On admission, August 26, the patient's temperature was 99 F., pulse rate, 80; respiratory rate 18, and blood pressure, 90 systolic and 60 diastolic. She felt dizzy when she was put to bed but slept well the first night. On August 28, she received two doses of acetylsalicylic acid for headache; after a perimetric examination, she complained of vertigo. On August 29, the headache was more severe and she vomited; acetylsalicylic acid and sodium bicarbonate did not give relief. On Aug. 30, a lumbar puncture was done about 9 a. m. Some time later, she vomited some yellow material. She sat up several times during the day. In the afternoon, she complained of severe headache over the top of the head and vomited. At 6:30 p. m., she was given 5 grains (0.324 Gm.) of amidopyrine which she vomited, and an ice cap was placed on the head. She complained continually of nausea. She died suddenly at 7 a. m., twenty-two hours after the lumbar puncture had been performed.

The patient was under the care of Dr. Thomas K. Davis, who made the following additional statement: "One has no grounds on which to consider the death as due to the lumbar puncture either as a certainty or a probability. I feel that it is only a possibility which cannot be excluded. The interval between the puncture and her death was approximately twenty-two hours. Sudden unexplained death after puncture in the presence of an expanding lesion, after an interval approaching twenty-four hours, is no more frequent than in the ordinary advanced

case in which lumbar puncture has not been done. I consider it only a fairly remote possibility that the puncture in this particular patient brought on her death."

From a study of the hospital record, I reached a conclusion agreeing with the opinion of Dr. Davis; but, in fairness to this study and owing to the fact that the patient's course differed so greatly from that of 199 others, it seems better to consider that death in this case was probably due to lumbar puncture.

SUMMARY AND CONCLUSIONS

This study of 200 cases in which diagnostic lumbar puncture was performed gave the following results:

1. In 94 cases of verified intracranial tumors, in all of which more or less well marked signs of increased intracranial pressure were noted, and in 62 of which the growths were supratentorial, the removal of a small amount of fluid by lumbar puncture did not give rise to any serious symptoms. Occasionally, as after any lumbar puncture, headache became more severe for a few days and, in one case, weakness and rapid pulse ensued for a few minutes.

2. In 106 cases in which a tumor was suspected, but the diagnosis not verified, 79 of which were supratentorial, there was one instance (G. M., reported in detail) in which serious symptoms followed the lumbar puncture and death occurred, possibly as a result of the withdrawal of spinal fluid. In the other 105 patients, symptoms of significance were not caused by the careful removal of fluid from the lumbar subarachnoid space.

3. None of the patients with verified or suspected infratentorial new growths, in whom lumbar puncture was performed before the diagnosis of expanding disease in the posterior cranial fossa had been made or suspected, developed any untoward symptoms after the puncture.

Although lumbar puncture should not be done in patients with symptoms of an expanding lesion beneath the tentorium, it is of interest that in 59 cases in this series, of which 32 were verified tumors in or around the cerebellum, serious symptoms did not follow the withdrawal of a small amount of fluid by lumbar puncture. In most of the 59 patients, the puncture had been performed before the diagnosis of a subtentorial tumor was made or suspected.

The study of 200 cases of tumor of the brain with increased intracranial pressure led me to conclude that in such patients there is not any danger from diagnostic lumbar puncture, if it is carried out with the patient in a horizontal position and with a needle of small caliber, and if no more than 5 cc. of fluid is removed. If the fluid is found to be under considerable pressure, it should be allowed to escape slowly, and the patient should always be kept flat on the back in bed for twenty-four hours.

Clinical and Occasional Notes

SPONTANEOUS CEREBROSPINAL RHINORRHEA

Report of a Case *

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The spontaneous discharge of spinal fluid from the nose is most often associated with tumor of the brain, and specifically with lesions that interfere with the normal circulation of the spinal fluid. In 1926, Locke [1] reviewed the literature on this subject and found twelve fatal cases in which autopsy reports were sufficiently complete to permit definite conclusions concerning the cerebral and cranial changes associated with this condition. Internal hydrocephalus was present in all the cases, and in eight of them it had resulted from obstruction of the ventricular system caused by a tumor; in two cases, the hydrocephalus was of the congenital type with rhinorrhea developing in early adult life after the closure of the fontanels and sutures, and in two cases the hydrocephalus was classed as of the adult type.

In these cases, in addition to the internal hydrocephalus and the increased intracranial pressure, an opening must exist between the intracranial cavity and the nose. In eleven of the twelve cases reviewed by Locke, such openings were clearly demonstrated in the cribriform plate of the ethmoid, while in the remaining case it was probable that a similar communicating channel had been closed by the exudate of a terminal meningitis. In seven cases, the communications were found simply connecting the cisterna basalis with the nose; in three cases, a direct communication between an anterior horn of a lateral ventricle and the nose was shown, and in the remaining case it was thought that a persistent ventricular lumen in the olfactory bulb was present, from which a fistulous opening into the nose had formed.

In 1899, St. Clair Thomson [2] reviewed the literature on this subject and reported a case. He found what he considered were twenty instances of this condition, grouping eight as certain and twelve as probable. All the authentic cases showed cerebral symptoms. In three of four patients that died, cerebral lesions were demonstrated.

Thomson noted that in five of the cases there was an intermission in the flow of the spinal fluid from the nose. None of the cases showed a complete cessation of the flow. Locke [1] reported a case of chronic internal hydrocephalus in a man, aged 21, who developed cerebrospinal rhinorrhea following a fall. The nasal discharge persisted for almost two years and then stopped spontaneously. An exploratory operation, following injection of air into the ventricles, revealed an extensive pachymeningitis interna that prevented a satisfactory exploration of the brain. Following the operation, the patient remained "in deep stupor or coma" for a period of about two months; he then suddenly improved, and the cerebrospinal rhinorrhea reappeared. A year later, the patient was reported as being

* Submitted for publication, Oct. 5, 1928.

1. Locke, C. E.: Spontaneous Escape of Cerebrospinal Fluid Through the Nose; Its Occurrence with Brain Tumor, Arch. Neurol. & Psychiat. **15**:309 (March) 1926.

2. Thomson, St. Clair: The Cerebro-Spinal Fluid: Its Spontaneous Escape from the Nose; Monograph, London, Cassell & Company, 1899.

mentally and physically better, without recurrence of the rhinorrhea. It is doubtful whether this case should be classed as one in which the discharge of spinal fluid has permanently ceased. Williams [3] briefly referred to a case of spontaneous cerebrospinal rhinorrhea demonstrated by him before the British Medical Association in 1901, in which the patient subsequently recovered and remained well for at least two years. With these exceptions, I have not found any instances of recovery from the condition when it has become established.

REPORT OF CASE

History.—On May 15, 1924, L. W., a white woman, aged 52, a widow, was referred to me for examination. She complained of a continuous discharge of clear fluid from the left side of the nose. Her father had died at the age of 78 from "Bright's disease"; her mother had died at the age of 62 from "some nervous trouble" and was mentally deranged for some time prior to death. One sister had died of heart disease; three sisters were living, and all were said to have some ill defined nervous disorder; one brother was living and was described as "a nervous wreck." Her husband had died three years before of paralysis. The patient had never been pregnant. The positive points in the past history were: measles, mumps, whooping cough and influenza among the infectious diseases, occipital headaches for many years, shortness of breath on exertion, mild nausea at times, a tendency toward constipation, hemorrhoids without bleeding, nocturia from five to seven times for some years, moderate leukorrhea and mental depression at times. Her usual weight was 180 pounds (81.6 Kg.).

The present illness began about one month before I saw her. There had been a sudden appearance of a discharge of clear fluid from the left side of the nose. This had been profuse and had continued day and night. There had not been any blood or mucus in it. The patient said that she had had a more or less constant headache in the occipital region for five years preceding this discharge, and that the headache had been neither better nor worse since the dripping began. She had not had any other discomfort, any dizziness or convulsions or loss of consciousness. She had not noted any muscular weaknesses or changes in vision.

Physical Examination.—The patient was rather obese, weighing 187 pounds (84.8 Kg.). She was 5 ft. 6 in. (167.64 cm.) in height. The skin was free from eruptions and abnormal pigments.

The eyes were slightly bulged, the left a little more than the right; the von Graefe sign was positive; the eyes converged normally, and there was not any sign of weakness in any of the extrinsic eye muscles. The left pupil was slightly larger than the right; both pupils reacted normally to light and in accommodation.

There was a steady drip of clear fluid from the left nostril. There was no obstruction on either side of the nose. The nasal mucous membrane was not swollen or congested.

The ears were normal. The upper teeth were artificial. Four lower teeth remained, all of which showed some degree of infection around them. The buccal mucous membrane was not inflamed. The throat did not show any signs of infection, and there was not any postnasal discharge. None of the superficial lymph glands were enlarged. The thyroid was not enlarged.

The heart was definitely enlarged to the left. There was no dulness behind the manubrium. The sounds were clear at the apex and the base, with a slight

3. Williams, W., in Allbutt and Rolleston: System of Medicine, 1909, vol. 4, pt. 2, p. 70.

accentuation of the aortic second sound. The heart action was somewhat rapid, but regular. The abdomen was normal. The extremities did not show edema. Tremor of the fingers was not noted.

The tendon reflexes were normal throughout. The Babinski and Romberg signs were negative. Kernig's sign was negative. Muscular weakness was not present.

Careful testing of all the cranial nerves failed to reveal any abnormalities. Specifically, the olfactory sense seemed unimpaired, the vision was normal in both eyes and the hearing acute on both sides.

The blood pressure was, systolic, 195; diastolic, 120. The pulse was 120 in rate and regular. The temperature was 97 (11 a. m.).

The urine (voided) showed: specific gravity of 1.010; albumin, positive; sugar, absent; a great many hyaline and granular casts, many pus cells and an occasional red cell.

The output of phenolsulphonphthalein was 65 per cent in two hours.

The examination of the blood showed: hemoglobin, 87 per cent (Sahli); red cells, 5,088,000, and white cells, 14,000. The differential count revealed: polymorphonuclear neutrophils, 82; polymorphonuclear eosinophils, 1 per cent; small lymphocytes, 10 per cent, and large mononuclears, 7 per cent.

A stool was negative for occult blood and parasitic ova.

The visual fields did not show constriction, and the color fields were normal. The eyegrounds showed slight haziness of the optic nerve heads with normal physiologic cups. The arteries were tortuous with prominent light-streaks and they compressed the veins on crossing. Exudates and hemorrhages were not observed.

Nasal Discharge.—The fluid from the left side of the nose was found to be dripping at the rate of 1.5 cc. in five minutes, or an estimated quantity of 432 cc. in twenty-four hours. It was clear and had a specific gravity of 1.005. It contained three cells per cubic millimeter, showed a faint cloud in the Noguchi test and gave a slight reduction of Fehling's solution.

The Wassermann reaction of the fluid and of the blood was negative.

Course.—On June 2, 1924, the patient was seen again. The fluid was still coming from the nose at the same rate as before. Changes were not noted in the general condition except that the blood pressure was, systolic, 235; diastolic, 140 (Baumanometer).

Repeated efforts were made to have the patient return for further observation, but nothing was heard from her until May 17, 1928; then she wrote that the drip from her nose had stopped about two months after I last saw her and had not reappeared. Her family physician reported at the same time that the blood pressure was still high, but that she seemed to be in good condition otherwise.

Diagnosis.—Cerebrospinal rhinorrhea; tumor cerebri (?); arteriosclerosis, and hypertension.

This case of spontaneous cerebrospinal rhinorrhea seems to be of especial interest because of the following considerations: (1) the complete cessation of the flow of spinal fluid from the nose for a continuous period of more than four years; (2) the absence of any demonstrable intracranial change other than the cerebral arteriosclerosis inferred from the observations of the retinal arteries, and (3) the association of the cerebrospinal rhinorrhea with extreme hypertension and the possible interrelationship of the two conditions.

PARERGASTIC REACTIONS AND REACTION TYPES: SCHIZOPHRENIA *

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Bleuler,[1] in 1908, substituted the name "schizophrenia" for the term dementia praecox. Kraepelin,[2] in 1896, had collected under the latter name a large group of cases which he designated in the fifth edition of his textbook deteriorating psychoses (Verblödungspsychosen) and had opposed them to a group of cases that tended to recovery; the latter he designated periodic psychoses, and later [3] manic-depressive insanity. This proposal of Kraepelin represented the most important step in scientific psychiatry after the beginning of the nineteenth century. The only really important step that had been taken before that time was the clear delimiting of the organic cases from the so-called functional insanities. Paresis had been recognized as due to a definite disease of the brain as early as 1798 [4] by Haslam, and particularly by Bayle, in 1822;[5] at the same time it became clear that definite alterations of the brain accounted for the arteriosclerotic changes of later life and the dementias of old age.

Esquirol,[6] as early as 1805, pointed out that there were idiocies or deteriorations which were not congenital but acquired. It was also known that certain acute cases of insanity ended in recovery, while others went on to "weakmindedness"; that furthermore some states of acquired weakmindedness occurred without any preliminary mental disease of the character of melancholia or excitement, and yet were not organic. For example, Griesinger,[7] in 1845, stated that cases of

* Submitted for publication, Oct. 25, 1928.

* This article was read as a lecture before the Yale University School of Medicine, Nov. 18, 1926, as part of a lecture course in psychiatry, psychopathology and mental hygiene.

1. Bleuler, E.: Dementia Praecox oder Gruppe der Schizophrenien, in Aschaffenburg, G.: Handbuch der Psychiatrie, Leipzig and Vienna, Franz Deuticke, 1911, pt. 4, sec. 1.

2. Kraepelin, E.: Psychiatrie, ed. 5, Leipzig, Johann Ambrosius Barth, 1896.

3. Kraepelin, E.: Psychiatrie. Ein Lehrbuch für Studierende und Aerzte, ed 6, Leipzig, Johann Ambrosius Barth, 1899, vol. 2.

4. Haslam, John: Observations on Insanity; with Practical Remarks on the Disease, and an Account of the Morbid Appearances on Dissection, London, Rivington, 1798.

5. Bayle, A.: Recherches sur les maladies mentales, Paris, 1822.

6. Esquirol, J. E.: Des passions, considérées comme causes, symptômes et moyens curatifs de l'aliénation mentale, Paris, Didot, jeune, 1805.

7. Griesinger, W.: Die Pathologie und Therapie der psychischen Krankheiten, ed. 2, Stuttgart, A. Krabbe, 1861; Mental Pathology and Therapeutics, trans. from the second German edition, London, The New Sydenham Society, 1867.

"acquired dementia may, of course, arise primarily—that is, without having been preceded by another form of mental disease, or by other cerebral disease of a different nature, as in the case of mental weakness of advanced life or premature decrepitude, primary atrophy of the brain, intracranial tumors, etc."

Nevertheless, chaos reigned in psychiatry during the middle half of the century; psychiatrists described cases that represented clinical pictures of their own experience. The one feature common to all the descriptions was that they represented clinical pictures only. Kahlbaum,[8] driven by a desire to describe diseases and disease entities that would follow the pattern of dementia paralytica, with a definite etiology, course and outcome, began, in 1863, to formulate his theories; between 1868 and 1874,[9] he established catatonia as a clinical entity, a disease characterized by stupor and deterioration. He was dominated, however, by the idea that all cases of this type followed the pattern of vesania typica and that in every case of catatonia there was first a stage of melancholia, and then of mania, followed by stupor, confusion and dementia. True, any one of these stages might be omitted in an atypical case, but this was an exception to the rule. Hecker,[10] in 1871, stimulated by Kahlbaum, separated off a group of cases under the name of hebephrenia. It was Kraepelin then, in 1896, who insisted that all these cases and many paranoid forms were subdivisions of one disease; that both primary and secondary dementia were due to the same causes; that the variety of manifestations was simply symptomatic of an underlying organic process, and further, that the outcome could be predicted from the beginning by a study of the symptomatology. The nature of the underlying process was unknown, but it probably was due to some endogenous toxin or metabolic disorder. He suggested the possibility that the sex glands were a seat of the difficulty, but did not present any evidence on this score. He defined this group of cases as occurring in youth and terminating in dementia, and as characterized by special types of mental disturbances: hallucinations; disturbances of attention through failing interest; incoherence and dilapidation of the course of thought; defect of judgment; formation of delusions; emotional dulness; reduction of voluntary activity, going as far as stupor; tendency to stereotyped movements and attitude; automatic responses to suggestion; catalepsy; unwillingness or inability to work, etc.

In this presentation, I shall avoid the error of Kraepelin, who presented his description of the disease by giving great masses of special

8. Kahlbaum: Gruppierung der psychischen Krankheiten, Danzig, Kafeman, 1863.

9. Kahlbaum: Die Katatonie oder das Spannungsirresein, Berlin, A. Hirschwald, 1874.

10. Hecker: Die Hebephrenie, Virchows Arch. f. path. Anat. **52**:394, 1872.

symptoms, without giving individual cases with their particular development. Instead of enumerating all the delusions or all the hallucinations that may possibly occur in this disease, I propose to study delusions and hallucinations in a setting that does not tear life and experience asunder into piecemeal symptom-categories, but leaves mental disease a part of the natural history of a life and its experience.

I shall present four histories, taken almost at random, in cases of mental disease diagnosed as schizophrenia in the Phipps Clinic of the Johns Hopkins Hospital. In case 1, it will be difficult to distinguish where faulty training and bad habits left off and the mental illness began.

REPORT OF CASES

CASE. 1.—*History.*—The patient, an unmarried man, aged 24, was brought to the hospital because he had been refusing to see anybody, would not eat and had outbursts of temper, with "weak spells." It was learned that he was the only son of a nervous, violent tempered father, who had died when the patient was young. The patient's mother, a kind hearted, nervous person, idolized the boy and spoiled him to the last degree. She could never say no to anything that he wanted or wanted to do. When the time came that some discipline had to be instituted, the boy went into tantrums. This method of behavior finally forced the mother to yield to his every whim and desire. When allowed to have his way, he was mild-mannered. He was overly attached to his mother; in fact, he slept in the same bed with her until he was 11 years of age. His tendency was to avoid other children and to keep his feelings and ideas to himself. While in the grade school and during the first two years at high school he did excellent work, but he failed to pass the examinations in the junior year. The family physician discovered that the boy had been masturbating to excess since the age of 13, and advised circumcision. This, however, plus the constant surveillance of the mother, did not stop the masturbation; over and over again he would promise to stop and then would break his word. He began to feel that his will power could not hold up against his desire. The mother's constant anxiety about this auto-erotism led her to try to frighten him by telling him that it would lead to insanity. He lost weight and his color became pasty and sallow. At the age of 18, he showed curious attacks of jerking, with fits of laughter. These attacks, for a period of six months, would occur from one to twenty times a day and were not accompanied by loss of consciousness. They gradually disappeared, and for the next two years he was apparently more nearly normal. When he was 20, however, it was noted that he wanted to be alone all the time and felt "a horror" when anybody looked at him. He said he was filled with "feverish energy." In the hope that a new environment would help, he was taken to northern New York and he did actually improve. However, on his return home he was tense and restless. One day while he was shaving the mother heard him call out, and when she reached him found him with his throat cut. While he was recovering in the hospital, he made another attempt to commit suicide by jumping out of the window. The following winter he had attacks while standing, in which he would become rigid and tense, and would stare for ten or fifteen minutes. At no time did he become unconscious, but these attacks were so automatic that in one of them he rolled downstairs. His appetite failed and he ate irregularly. He tried to seclude himself in a dark room. Because of

the recurrence of the tantrums of marked temper, he was taken to a hospital for mental disorders. During his twentieth year, he had attempted to study chemistry at a university in his home city. From that time, he felt that he could "reach out and control nature" through his scientific studies.

Examination.—When first seen at the hospital, the patient was unable to look directly at the physician. He appeared tense and talked with head and shoulders bent forward. He smiled and grinned in a way that did not carry conviction to the observer, and complained of weakness. He said that this was because his nerves were "knotted up." He explained that he could not eat solid food. "It jolts the base of my brain," he said. He refused to sleep in a room without a light. He avoided contact with other patients, and rarely spoke unless spoken to. His talk reverted continually to his nervousness; when urged to talk he would say that his nerves would soon relax, and then he would feel normal and be able to talk and "influence other people." When asked about his mood, he said that he "was in a life and death struggle, but that it wouldn't last more than twenty-four hours." He talked about peculiar experiences, saying that he had had wonderful sensations; that he was "like Rider Haggard"; that he had been growing mentally but not physically, and that he had lost an inch in height because of the terrible things he had been through. "My bones have just dried up." He explained that the "mind controlled the operations of the body," and made the assertion, "I have stopped the beating of my heart for a minute." "Once my blood turned a pale pink."

He knew where he was and to whom he was talking; his memory was excellent, and there was no defect in his knowledge of general affairs. He knew that he was sick, but insisted that it was all due to his physical condition. Actually, physical and neurologic examinations did not show abnormality.

Course.—In the next few weeks, a marked change was not observed. He was forced to eat regularly, but all efforts to get him to do any kind of work were unavailing. He was "too nervous"; or "would be able to in a little while," but that time never arrived. Frequent attacks of trembling appeared, which he admitted were accompanied by sex feeling. He continued seclusive and said that he wanted to withdraw from the world.

A note made five years later at another hospital said that the patient referred to his "innate ability to understand the problems of life and immortality; to remain young for a thousand years through the effort of his own will"—thus "keeping the elements of the body in motion which is the basis of all life." He was unable to interest himself in any occupation and, though clean, did not show any interest in his personal appearance. A year later he escaped from this hospital and, eluding the guard at the top of the Washington Monument, jumped to death.

Comment.—This is a typical story which I have purposely kept entirely in terms that represent life situations and life experiences. An analysis of the facts reveals, in the first place, extremely faulty early training and discipline: a nervous, overanxious mother lavished undue affection on him and attached him to her by being with him constantly and by not giving him the chance to develop outdoor activities and to do manipulative work; slept in the same bed with him almost to the time of puberty; indulged him to the point that he learned nothing of self-control, and then allowed him still further to control his surroundings

through tantrums of temper. When puberty and sex problems arose, he had none of the normal alternative outlets of boyhood, nor had he been able to establish through training any of the control that is necessary to combat momentary desire. He was told repeatedly that masturbation would lead to insanity, and was scolded and tormented on this point. He felt that his will was growing weaker and weaker, and he finally reached the place where he thought others could read the habit in his eyes. He secluded himself, and because he did not have wholesome interests turned more to daydreaming and rumination. In this ruminative attitude, it required less and less stimulus to set off sexual feelings. Ruminative fantasy led to the solution of his problems through philosophic speculations about the control of the body through the mind. Mere thinking started sexual reactions that appeared as attacks of trembling, which, according to the patient's own account, were sex equivalents. His preoccupation with philosophic speculation and the horror of uncontrollable sexuality made work and concrete interests unsatisfactory so that there was a further decline in the concrete activity that might have led to a correction of the distorted daydreaming. Contact with the concrete real world lessened; activity became more and more automatic, and was dominated by speculation and odd uncontrollable impulse and wilfulness. He showed a definite deterioration of interests and habits, amounting to dementia.

For diagnostic and prognostic purposes, this is a typical example of dementia praecox in the kraepelinian scheme. Kraepelin, in his textbook descriptions does not give any case material, but only the composite pictures as they appear from his method of examination. If one turns to the sixth edition of the textbook and compares this case with Kraepelin's description, one will find that it conforms in all important respects. In this description (1899) he said that he could not doubt that "in this disease we deal with a severe damage to the cerebral cortex, which at best is only capable of partial repair. Although the manifestations of the disease that are observed during its course show a great diversity—in fact, so much that it is often difficult to recognize any inner connections—nevertheless, certain fundamental disturbances are encountered which persist after the disappearance of the accompanying symptoms. This is especially true of the terminal state."

Kraepelin's general description of the particular symptoms is as follows: Comprehension and orientation are intact; consciousness is clear, although it may be clouded in excitement and in stuporous states. Memory is undisturbed. Retention is usually good. On the other hand, illusions may dominate the picture, especially at the onset, and hallucinations, especially of hearing, are usually prominent in the beginning, although they tend to disappear. Their content is usually senseless and without connection. Attention is disturbed and this is dependent on

failing interest. This is most marked in the stuporous states. With the passing of the stupor an undue curiosity may arise, only in turn to be restricted by a tendency to negativism. The stream of thought sooner or later suffers. This is manifested in a looseness or slovenliness of thinking. It may be manifested in the milder cases only by a heightened distractibility, a jumping from topic to topic or an excess of talk or circumstantiality. In more severe cases, it goes on to complete confusion of talk with loss of connections and the coining of new words (neologisms). There is also a tendency to stereotyped utterances and frequently a tendency to rhyme, to make use of senseless "Klang" associations and punning. Judgment is usually poor in the formulation of new plans and the interpretation of experiences; proper conclusions are not drawn from given facts. At best there is only a superficial recognition of their illness by the patients without any true understanding. Delusions are common—transitory or persistent—at the onset. These may be hypochondriac, persecutory or moral; later, delusions of grandeur come to the front. As a rule, the delusions have a senseless or fantastic coloring, due to the rapidly developing psychic weakness. The delusions tend to disappear, except in the paranoid forms. In the emotional life there are profound disturbances. At the onset there is often a sad or anxious mood; more rarely there are states of hilarity with uncontrolled laughter.

More important, however, is the universal appearance of emotional dilapidation or apathy, which is one of the fundamental features of the disease. There is an indifference to former relationships, friends and family, and a loss of satisfaction with work. The patients do not any longer feel happiness or sadness; they become dull and emotionless. They are often indifferent to pain or injury. With this there may be definite irritability. In the field of general behavior and activity they show marked disturbances. There seems to be a general reduction in the will to do; they lose the drive to work, neglect obligations and sit about aimlessly. At the same time there may be a great deal of motor activity, which is apparently an aimless expression of inner tension. This may result in impulsive, violent actions, without any apparent purpose and without any consideration of results. This inability to suppress impulses appears in the states of stupor as well as in the excitements. Blocking appears primarily in the stupors; each impulse is immediately wiped out by a stronger one in the opposite direction. This gives rise to negativism with resistance against change in position and against food and clothing; retention of urine and saliva, etc. The sudden impulses may arise and not be repressed by negativism and then one observes repeated activities such as stereotypies of movement and positions, verbigerations and mannerisms.

With the severe injury of the will, the destruction of the inner motives, desires and restraints, there arises command automatism and increased suggestibility. Under this heading come catalepsy, echolalia and echopraxia.

On the physical side, one sees epileptiform convulsions, rigidities and contractions, exaggerated knee jerks and an increased mechanical excitability of the muscles and the nerves. The pupils are frequently dilated, and there is frequently a shifting pupillary inequality. There are vasomotor disturbances—cyanosis, circumscribed edemas, undue sweating, etc. The bodily temperature is frequently lowered, and the menses are suppressed.

In discussing the etiology, Kraepelin pointed out that in 70 per cent of his cases the heredity was defective; in only 20 per cent of his cases, however, was there any peculiarity in the patient prior to the outbreak of the disease, such as irritability or seclusiveness. Kraepelin argued that some specific organic cause must be at work in the brain to account for the outbreak of such a disease in a healthy person, even when one grants the possible hereditary tendency to mental disease.

The fundamental symptoms, then, can be summarized as follows: (1) deterioration of interest; (2) looseness and slovenliness in the stream of thought; (3) emotional dilapidation going on to indifference, with the appearance of impulsive activity and automatisms; (4) a failure of the will to do, and negativism.

The hallucinatory and delusional experience is likely to be transitory and is dependent on rapid mental deterioration.

The patient in case 1 presented the following group of symptoms: marked deterioration of interests, dilapidation in thinking, definite indifference with occasional outbursts of anxiety and vague fear, the feeling of automatic activity and the formation of expansive delusions. Nowhere in this kraepelinian scheme does one find any mention of the life situations and experiences that I have described. The delusional material and hallucinatory experience, according to Kraepelin, are senseless symptoms, which are dependent on hypothetic alterations of the brain; the failure of the patient to keep alive in his habits of work and contact with the world are mere evidences of the effects of a toxin; opinion as to the source of this toxin rests wholly on conviction and not on even remote evidence.

Bleuler, the most outstanding proponent of the kraepelinian doctrine, found it necessary to modify the conception of Kraepelin in some essential points. Working with his own scheme, Kraepelin found that prognostications were uncertain; even more important, he did not have satisfactory diagnostic criteria. For example, the diagnosis of dementia praecox in Kraepelin's clinic was made, at various times, in from 8 to

52 per cent of the admissions and later again in but 18 per cent. The growth of the freudian psychology, which was forcing the view that psychologic deviations do not come out of a clear sky but are understandable in terms of life experiences and conflicts, had its influence on the study of this group of cases. Bleuler was quick to recognize the importance of this newer psychology and, in 1911, in his monograph on dementia praecox,[1] he suggested important modifications of Kraepelin's views. Bleuler still rigidly adhered to the notion that a primary organic process in the brain was the basis for the disease and that this process imposed itself on an organism free from damage until the disease occurred. His own studies and those of his pupil Jung, who had been an appreciative follower of Freud, made clear that the psychologic manifestations and activities that had appeared senseless to Kraepelin actually have their origin in the life experiences and conflicts of the patient. His strong conviction, however, based essentially on the unalterability of the process once it became manifest, still forced him to the belief that there must be a primary organic alteration as a basis on which these psychologically determined symptoms developed. Bleuler divided the symptoms of the disease into two groups:

1. Fundamental Symptoms: These are the expression of the organic part of the process and constitute that part of the disease that remains as a residue after the acute phases of the disease have passed by. To this division belong the following groups of symptoms: (*a*) A particular disorder of association in which "the threads that lead our thought are broken, first in one direction and then in another, so that often the thinking is rendered illogical. Unaccustomed connections are made, condensations occur and there is a tendency to stereotypy, poverty of ideas, forced thinking, confusion and blocking." (*b*) Disorders of affectivity represented primarily by complete indifference, but at the onset frequently by oversensitiveness, and more especially by a failure of the proper depth of affect and its proper modulation, resulting often in whimsical, bizarre emotional responses, and often in inadequacy of response. This affective disorder is formulated by Bleuler as rigidity of affect (Affektsteifigkeit). (*c*) A tendency to ambivalence in effect, activity and intellectuality. This ambivalence makes a tendency to emotional, intellectual or motor activity and its opposite appear at one and the same time. These three groups of symptoms are fundamental, but are disturbances of the simplest order of mental functions.

Disturbance of the combined functions gives rise to a second order of fundamental symptoms which are grouped as autism or autistic thinking. This represents a disturbance of the patient's attitude to the outside world of reality. He loses contact with the outer world, and lives in a world of his own wishes and their imaginary fulfilment. This may become to him the world of reality. Attention may suffer in con-

sequence; apparent loss of will (abulia) with apparent laziness and neglect follows, and there is a loss of the integrity of the personality.

The essential schizophrenic dementia shows in the disorder of thinking and feeling, but its particular coloring is dependent on the secondary symptoms; ultimately, the behavior and the activity of the patient are stamped with loss of interest, initiative and purpose.

2. Accessory or Secondary Symptoms: These arise on the basis of the primary symptoms, but are colored by the material of the psychologic complexes of the individual patient. They consist of: (*a*) Hallucinations, principally of hearing and of bodily alterations. (*b*) Illusions. (*c*) Delusions, most frequently of persecution, but also of poisoning, of grandeur, of erotic aspiration, of hypochondriac notions and of reference. The delusions usually do not have any logical consistency, even within themselves. The splitting of the personality (as the name schizophrenia suggests) is clearly seen in the delusions, because they are so frequently rejected as not having anything to do with the person himself. (*d*) Falsifications of memory on the basis of delusions and hallucinations.

Speech, as the result of the primary symptoms of blocking and the poverty of ideas, and as a result of the secondary hallucinatory and delusional symptoms, may show marked alterations: overtalkativeness, mutism, verbigeration, mannerisms, neologistic talk through the process of condensation and symbolization and, finally, a word salad.

Bleuler suggested the change in name because the process frequently does not begin in youth and need not terminate in dementia. He pointed out further that it may halt anywhere in its course or regress in part; according to his ideas, however, it never goes on to complete recovery. This name is gradually coming into accepted usage.

Further cases may be cited to illustrate the mechanisms reported by Bleuler.

CASE 2.—One evening I was called to the accident department to examine an English sailor, aged 21, who had been taken forcibly out of the bay and resuscitated by the pulmotor after an apparent attempt at suicide. When first seen, he was lying quietly on the bed with an ecstatic smile on his face. When asked what the trouble was, he said he had tried to swim to the sun but everybody was hindering him. His story dated back five years to a time when he had fallen in love with a young girl. They were engaged for two years, and then one evening she invited him to her house and made sex advances. This shocked him, and shortly after, when he received a vulgar letter from her, he dropped her. He then went to sea. Occasionally, he had promiscuous sex relations, but this always led to a great deal of remorse and disgust. Within the two years preceding his examination he masturbated rather frequently; he felt that all his sex activities were sinful and were put in his way by the Lord to tempt him. Five months before his "swim to the sun," he came to the conclusion that he needed the help of a woman to keep him from his sinful life, and he wrote to the same girl asking her to meet him. When she came she was drunk, and an

agreement could not be reached. He saw her again three months later, but she was cold to him, and told him that she was in love with some one else. After that he had become worried and tense and suffered a great deal of sexual desire. He masturbated frequently and worried whether he was not damaging his sexual power. A month later, after landing in America, he wrote to the girl making a formal proposal of marriage, but immediately began to worry as to whether he could support both a wife and a mother. He decided to try for a better job to earn more money and began to study nautical matters. He wondered further whether he was fit for marriage, on account of his sinful life of venery, and felt that he must be purified before marriage. He went to the ship's physician to try to get medicine to help him in meeting the problem of masturbation.

In the weeks preceding the break, he had a heavy cold, and then, two days before the swim, his head suddenly cleared and he felt happy and cheerful. With this increased happiness, he felt that he could do things that he had been unable to do before; his voice became a fine singing voice, and he sang a great deal. His restlessness continued; he could not sit through a movie, but "got all shaky inside." All his feeling about having been tempted by God recurred, and he felt ashamed of his sin and determined to clear his conscience, keep clean and help others.

He had read on a calendar, "Never let us make a shadow by turning our backs on the sun." On November 3, this came into his head as a guide to salvation. It seemed as if everybody was defying him to keep him off the straight path. He went ashore and tried to get a job on another ship, but was told that they did not have room for him. He then decided that he must seek his own salvation alone, and the words on the calendar seemed to him to mean "Follow the sun." The sun seemed to move and lead him on. He walked toward it, climbing over everything in his path. He clambered over the scenic railroad at River View Park and reached the beach. Everything seemed perfectly silent and pitch dark except the sun. He felt as if the world would stop if he did not reach it and that he alone could save the world. He jumped into the river and swam toward the sun. Across the face of the sun he saw the crucifix. Everything seemed to combine to stop him. Black birds flew over his head to lead him astray. The ships in the harbor got in his way. Voices behind him told him it was impossible and mocked at him. He felt that these were the voices of evil bent on preventing his salvation. The sun represented the highest happiness with the girl—salvation and love. It protected him from harm, and he heard the voice of God from the sun calling him.

When 500 yards from shore, he was fished up by two men in a boat and brought ashore despite his resistance.

When questioned about this whole experience, he said that he felt as if he were in a new world altogether. It all seemed to him like a biblical experience. The sun, which had been at first a goal, soon seemed to command him and then everything forced him to go on. He became automatically driven by the forces of the sun, God, love and the crucifixion.

Comment.—In this there is a sort of religious ecstasy; the patient abandoned the real world, in which circumstances made him too unhappy (a disillusionment about the woman he loved; the longing for a wife; the financial burden of his mother's support, which made marriage too difficult; the jolt of not being able to get a better job, and, on the other

hand, an inability to reconcile his religious views with the uncontrollable sexual desire within him), and took flight into a world of fantasy where at one fell swoop he purified himself and united mother, God and human love. This was all in a setting of ecstasy. It was the sudden creation of a world of his own in which all conflicts and difficulties were solved—in Bleuler's terminology, an example of autistic thinking and creation. The outer world was rejected or disregarded until his plans were interrupted; even then he still felt that he could live on in this new creation.

"The feeling of being forced and driven on" should be especially noted; it represents a type of reaction which to me is the most important feature of the schizophrenic picture. It should further be noted that this psychotic episode is an episode in a human life, and that the psychosis is part of a causal chain that becomes intelligible only when one knows the experiences and the conflicts of the patient prior to the outbreak of the disorder.

CASE 3.—A woman, aged 56, was brought to the hospital because she communicated with spirits and with her dead husband. Her mother had died in giving her birth and three years later, when her father remarried, she was adopted by an aunt, who raised her with great strictness and prudery. She made satisfactory progress at school, and was considered bright and normal in her contacts with other children. At 21, she married a naval officer, and a child was born one year later. Her husband petted and spoiled her, and she was devoted to him. He died nineteen years after the marriage. She thought of remarriage, but could not tolerate the thought of other men's caresses. She therefore cut herself off from associations with them, and went to work to support her daughter, working first as a seamstress and then as a government clerk. At the age of 46, she had a nervous breakdown, said to have been due to overwork. She was tired and weak. She rested for seven weeks and then returned to work, but quit after three months on account of trouble with her eyes. For the next four years, to the age of 50, she merely visited among friends. At the age of 54, she began to have so-called heart attacks in which she had difficulty in breathing, and palpitation. Six months before admission, she had an attack in which she felt numb all over. At this time, she received "a message" from her father, saying that she was going to die. During recovery from this attack (physical examinations did not reveal any cardiorenal or respiratory disease) she began to get communications from her dead husband saying "I'm glad you're getting better—I can help you—I am going to give you advice." A month before admission, she woke her daughter one night to tell her that she had received "word that a burglar was in the house" and asked the daughter to look for him.

The patient's only daughter had felt a growing dissatisfaction with her mother, and when the daughter married, the estrangement became more marked. The persistent physical symptoms—heart attacks and gastro-intestinal complaints—instead of arousing sympathy from the daughter, only irritated her the more. The patient became more and more dissatisfied as she realized that her isolation and loneliness were unalterable. Her dead husband continued to speak to her and advise her what to do. She took up automatic handwriting, which gave

her further advice through "thought transference." A week before admission, she began to hear definite voices, but different ones in the two ears. They were spiritual voices, a black devil in one ear and an angel in the other. The devil wanted her "to think adultery and say obscene things," and she "felt forced to say bad words." This voice also said, "Don't go to church on Sunday because you will blaspheme." The angel Gabriel, speaking in the other ear, told her she was pure. On the other hand, the voice of her husband said: "Don't talk to the voices during the day time because they will only deceive you. I will talk to you only at night." She was reading a chapter in the Bible at the request of her husband's voice and then, remembering that it was day time, became convinced that she had been deceived by the devil in disguise. She opposed coming to the hospital for fear that she should have to give up her ideas of communication with her husband and she did not want to discard so pleasant an experience.

In the hospital, the patient appeared normal in behavior, activity and social bearing. She was willing to give up all the voices but could not abandon the communications with her husband. She said that she had felt forced to do the automatic handwriting and that someone was trying to make her do the things she was doing. She thought the voices might be due to a tired brain. She said that the first voice and communication had come after the use of the "ouija board." At times, she said, the voices were more like thoughts, but at other times they seemed real. After two weeks in the hospital, she suddenly said that the whole experience was imaginary and asked for permission to leave. The night before her departure, she had an attack of palpitation of the heart; she said that her heart was all wrong and that all her blood was in her head. The next morning, she was talkative, irritable and demanded discharge. Her daughter reported that the patient had told her that she had told the physicians that she regarded her experience as imaginary only in order that she might be released from the hospital.

While in the hospital, the patient gave an account of her sex life, which throws light on the illness. She said that she had not been like other children, but had had to do "the proper thing" always. She was not allowed to learn to dance and did not lead a "young life" with boys and girls. She prided herself on having never told a lie and on her pure-mindedness. She disliked the whispered vulgarities of the girls that she knew and refused to do any clandestine kissing and hugging in darkened rooms or behind doors. She could recall that at the age of 10 she had had a revulsion of feeling at any masculine caresses. She did not have any sex urge, she said, and she regarded her marriage relations as having been "sweet and holy." She said that probably if she had had more sex desire she would have married again.

Comment.—The psychosis in this case was of gradual onset, at first with feelings of fatigue and weakness, later with eye strain and then abdominal and gastro-intestinal complaints. With growing dissatisfaction at a lonely life without affection, and a growing estrangement from her daughter, she began to have communications of comfort and advice from her husband; then she felt forced in her activity, and heard her conflicting desires and wishes as good and bad voices in her two ears. This again shows the creation of an autistic world in which the problems of life are solved, albeit with difficulties, because part of the wishes of the patient are not acceptable to her official personality. Again there is

the evidence of "feeling forced" and being passive in a fantasy world that rejects the experiences of the real world.

I may again point out how this personality could come to this type of solution. She was raised in a rigid, puritanic atmosphere, without social outlets; there was the setting at a preadolescent age of a sex pattern negative toward men and the fixation of this by prudish over-insistence on pure-mindedness. There was no opportunity for breaking down this pattern through the normal activities of play and courting, because she was taught to reject all such activities as "dirty." Her husband, instead of developing her into adult womanhood in her sex relations and in her outlook on the world, petted and spoiled her, and she lived through her marriage without much sex feeling, priding herself on the lack of desire and on her pure-mindedness. With the hard work of earning her living and the loneliness of her life, she developed hypochondriasis and finally gave up work. The possibility of remarriage occurred to her, but the physical side was revolting and she hid behind the memory of her husband's caresses. When the illness failed to command the sympathy of her estranged daughter, she was ready to convert the ruminations about her devoted, indulgent husband into reality. The ouija board offered a ready instrument (surrounded as it and similar devices are with mystical, half-earnest, half-joking credence) for the translation of her desires and wishes into reality. Automatic handwriting, which, though psychologically more automatic, still has some social acceptability, was the next move. The continued comforting communications from her husband must have led to more rumination about him, with a recrudescence of vague sexual fantasies—these, in turn, being rejected as incompatible with her purity of mind and chastity. When desire, born of loneliness, discontent and rumination about remarriage and old relations of affection with her husband became too insistent, it was rejected in this setting of mystical experience and was projected as not a part of herself—it appeared as messages that were at one time like thoughts and at other times like voices, depending on whether desire or respectability was uppermost. This situation was regarded by her as automatic, so that she felt forced to do things and to think things: another way of her saying that she was not responsible. To my way of thinking, this constitutes a possible chain of psychologic and experiential events needing no further explanation than this recital and an understanding of her personality, her early environment and the gradual substitution of fantasy, through the ouija board and automatic writing—instruments that are so acceptable socially.

One of the symptoms advanced by Bleuler as fundamental, and mentioned in the résumé already given, is disturbance of association—especially the blocking or the sudden interruption of the stream of thought or talk either by counterasserting impulses or ideas or by an

inability to continue the line of thought or talk. This, he holds, is evidence of the organic nature of the process because it seems to be a type of disorder that does not undergo repair and that leads to deterioration, when it exists in high grade. To understand this type of thinking, one must observe it in its simplest form, before it is overlaid with complexity. One learns about all the processes of disease through the study of the early lesions and by experimentally reproducing the disease so that the beginnings may be seen. The next case shows a patient responding with stupor to a difficult situation and incidentally gives a clue to the origin of the difficulty of association, which to Bleuler is so definite an evidence of organic alteration and to me is a mode of biologic or psychobiologic behavior; a mode, which is known as a normal reaction and is abnormal only when more dominating modes of behavior are wiped out and more primitive, undisciplined ways of response are released.

CASE 4.—A civil engineer, aged 34, was brought to the hospital because he was so nervous, suspicious and irritable. This change had appeared on July 4, two weeks before the date set for his marriage. He had been engaged for three years and had known the girl for fifteen years. During these two weeks, his friends had joked with him about his coming marriage; they had told him that they were going to put counterfeit money in his baggage and would then put the authorities on his trail. During the same period, the patient was worried over the difficulty of securing a proper passport, because it was hard to get a birth certificate in Pennsylvania, which had not had any consistent registration of births when he was born. He also began to wonder whether his sexual indiscretions of the past—masturbation and occasional illicit intercourse—had not made him unfit for marriage, and whether or not he ought to marry. Whenever he met the young woman, he was nervous, tense and suspicious. He told his friends that the authorities were after him and that he would not have enough money to bail himself out. He feared that a terrible disgrace would befall them all. He was taken to his home and, though silent and depressed, was in good touch with his surroundings. He was still, however, apparently puzzled about the passport. He asked his mother if she was really his mother and if his father was really his father. He appeared reassured by what his mother said on this point, but continued to be irritable, sullen and suspicious.

When brought to the clinic, he answered questions relevantly, but refused to allow the examination to be completed. When personal experiences were discussed, he admitted that he was depressed, that he heard voices and that he thought people might be against him. Two days later, he suddenly gave up his suspicious attitude and told how he had had suspicions on his way home, that he had been "experimented on," and that the "noises of the train were made to annoy him." He had thought that the babies he had seen on the way were displayed to see whether he would think they were his. The following day, he made a confession of all his past misdeeds so that he "could start life over again." He told of masturbation, drinking, smoking and illicit intercourse. He had had intercourse four times in the previous two and one-half years.

A lumbar puncture occasioned some discomfort, and the patient again became quiet and uncommunicative. A slight fever developed (there was a slight exacer-

bation of an old tuberculous process), which fell to normal within two days, but the patient became even more uncommunicative and lay stiffly in bed with fixed staring eyes; he refused to void or defecate, and would neither eat nor be fed. The following day he again began to talk and said he had not been talking because he thought he was supposed not to: "I was trying to assume the attitude of an infant so that I could begin life over again. I thought your sending me downstairs meant that I was to leave all my old life and its sins upstairs. I tried to put myself in a frame of mind to be born again." After this statement, he relapsed into stupor and remained in stupor for about a month. Toward the end of this month, he began to smile occasionally and then to go about attending to his needs. Finally, he began to talk. He discussed his experiences with great reserve and reluctance. To questions concerning impersonal topics, he would give prompt and intelligent replies, but when personal topics were touched on, he either remained completely silent, tending to drift off into daydreaming and inattention, or he would answer evasively and vaguely. For example:

Will you tell me why you didn't talk for so long a period?	Perhaps because I couldn't, Doctor.
Did you try?	No answer.
Do you remember thinking you might be arrested?	Some things I can't remember.
Yes, but do you remember that?	I don't see any reason why I should have been arrested.
Of course not, but you did say something about it.	I don't remember that; I was nervous.

Even when the patient's activity seemed normal, the pushing of personal topics still resulted in a drifting off into generalities or a complete inability to answer. After some weeks, he was able to discuss matters a little more freely and told of worry over an attack of gonorrhea that he had had four years before and which he feared was still present. He also said that he was not sure that he loved his fiancée enough to marry her. However, the patient, although he knew he wanted the engagement broken, could not be brought to a definite decision about the matter and the woman finally had to break it. He was much relieved by this.

A review of his past history brought out the following facts: He was an only child, reared by a religious father and mother, who instilled into him the strictest puritanic ideas. The mother was a nervous, high-strung woman, and her whole family was of the same sort. At the age of 5, he began to stammer, and this led to great difficulty in his contact with other children. He became shy, and tended to withdraw from the usual play with other children. At 16, he was forced by an unwise teacher to recite before a large class and his embarrassment was so great that he refused to continue attending this school. A year at a school for stammerers helped him so that he was able to enter an engineering school and to graduate at the age of 21. He felt, however, that people always pitied him because of his defective speech, and when people looked at him he was abashed and thought that they were being critical of him. He was devotedly attached to his mother and rather resented his father. When, years before the present illness, he became worried, nervous and depressed over a financial loss, he sent for a girl of whom he was fond. The mother disapproved of this girl, and the patient promptly dropped her. He admitted to us at the hospital, however, that he was fond of this girl, but had not persisted in his attentions because his mother did not want this girl, while she did approve of his fiancée.

Comment.—This patient again, according to the classic nosology of Bleuler and Kraepelin, was undoubtedly a schizophrenic. He had the fundamental symptoms of association disorder and autistic thinking, delusions of reference and of persecution, stupor, etc. The case presents, however, a chain of life events with conflicts arising out of a puritanic background; an undue attachment to the mother; domination of his emotional life by the mother, even to the selection of a wife; an unduly retiring sensitiveness, bred of consciousness of defectiveness of speech, and an inability to accept the forcing drive of sexuality because of the puritanic background. These influences, on account of his sensitiveness, could not be corrected by associations with companions. The only correction was a stubborn pride in work and a desire to succeed in work. With the closing-in of a marriage, the patient found himself trying to dodge, with worry over past sinfulness and feelings that he was not worthy of marriage. He probably wondered if he had been correct in accepting his mother's domination, and when he had difficulty about his birth certificate he asked whether she was really his mother—as if to say that he could avoid her domination if she were not really his mother; then, when he was trapped, he tried to return to the attitude of a child and wanted "to be reborn and start all over again." This is not my statement but that of the patient.

THE VIEWS OF ADOLPH MEYER

In spite of the continued efforts of Bleuler, Kraepelin and their pupils to see these cases as organic, convincing evidence to date has not been produced, either anatomic or chemical, which gives any clue to an underlying physical or chemical process. Every objection to the organic hypothesis, however, has been met with new hypotheses to explain away the discrepancies. Objections have been forthcoming from several directions, and it is worth while to discuss these in order that one may secure a clearer comprehension of the problem. Meyer,[11] in 1903, in a paper on the "Neurotic Constitution," and later Hoch,[12] in 1907, were able to show that a large percentage of the cases in this group present a definite type of retiring, seclusive personality. This, Hoch called "shut-in"—i.e., a tendency to seclusiveness, to diffidence, to shyness and to withdrawal from people and an inability to discuss affairs, etc. More recently, Kretschmer—still, however, dominated by the desire to find a physical basis for mental disease—described special types of physical constitution in which dementia praecox, or schizophrenia, develops. He found that long, thin people are more apt to

11. Meyer, Adolf: Neurotic Constitution, Am. J. Psychol. **14**:90, 1903.

12. Hoch, A.: A Study of the Mental Make-Up in the Functional Psychoses, J. Nerv. & Ment. Dis. **36**:230 (April) 1909.

constitute the group of schizophrenics, and that the short, rounded ones are apt to predominate in the group with manic-depressive psychoses. The constitution of the prepsychotic groups, he found, is also divisible into long, thin schizoids and short, rounded cyclothymics. The mental correlates of these types, in the schizoid group, are the shut-in types of personality with little affective response to the outside world of things and people; whereas, in the cyclothymic group, they are the types in more affective rapport that show fluctuations of mood corresponding to variations in events in the outside world.

Bleuler, accepting these types, changed the name of the latter group from cyclothymic to syntonic, because he preferred to stress the affective rapport rather than the undue variations of mood. Now, Bleuler was forced, in his acceptance of these two types, to admit that an exaggeration of the normal schizoid mentality may lead to a picture that is near to, or indistinguishable from, schizophrenia, though it runs a different course. In his unwillingness to renounce the concept of a disease entity, he presented two hypotheses. Special cases are, for him, symptomatic schizophrenia, in the sense proposed by Bonhoeffer in his study of deliria; i.e., in these cases the picture resembles schizophrenia due to cortical disease, but the condition does not follow the same course; the symptoms only simulate the disease caused by the hypothetic destruction of brain. Furthermore, he distinguished a new group of cases, which he called schizopathic, in which, on the basis of schizoid personalities, there develop symptoms which are not present in the same degree as in real schizophrenia, yet which present a picture much akin to that of true schizophrenia. Further, he had to assume the imposition of this special disease process on the schizoid type of personality, and then, with its exaggeration (schizopathy), he got real schizophrenia. To explain the appearance of schizophrenia when circumstances precipitate the attack, he invented the concept of latent schizophrenia—i.e., the disease is present as an old scar, which is reinflamed either by some toxin or by fever. This position was forced on him by the precipitation of schizophrenias from the experiences of the war and the occurrence of the disorder after or during physical diseases or toxic insults (alcohol).

Still another difficulty was presented. Although the textbook descriptions give a dogmatic picture that should make diagnosis and prognosis easy, in actual fact the individual cases offer the greatest difficulty as to both diagnosis and prognosis. Constantly, one sees a mixture or fusion of manic-depressive and schizophrenic symptoms, and the outcome in deterioration or cure is known only after the case has run its course. I shall take occasion to discuss the diagnostic difficulty later, but Bleuler's solution uses his constitutional types to

good purpose on this score. The schizophrenia arises in a syntonic (cyclothymic) personality, and the secondary symptoms, naturally making use of the particular personality and its situation, color it accordingly with symptoms like manic-depressive symptoms.

With one more criticism of Bleuler, I may proceed to a statement of our position in Dr. Meyer's clinic. This criticism of Bleuler is not meant to detract from the profound contributions he has made to the subject. His descriptions of the actual symptomatology of the disease, his analysis of the psychic aspect of schizophrenia, the autistic thinking, his theories of negativisms and ambivalence and his great clinical acuity will stand as permanent monuments in psychiatry. My criticism is directed solely against his refusal to accept life events and situations as scientific facts of the same order of efficacy as alterations of the brain and toxins. In one of his later publications, "The Differentiation of the Physiogenic and Psychogenic in Schizophrenia," [13] he gave a clear picture of the fundamental and the secondary disorders, and one would be glad to follow his prognostic criteria if he had spoken of malignant and benign symptoms. It is true that all symptoms classified as primary are apt to be evidence of profound lasting alterations of the personality, but these immediately became for him organic or physiogenic. His essential honesty put him in a dilemma, because it is precisely "in the chronic, clear schizophrenias that secondary symptoms of fixed delusions with psychogenic hallucinations and falsifications of memory and autism are in the foreground." Bleuler therefore had to assume that these psychologically determined manifestations, while not primary symptoms, are nevertheless physiogenic. The argument, then, would run something as follows: primary symptoms are evidences of organic disease—and are therefore not alterable; all unalterable chronic symptoms must then be organic.

Now what can be opposed to the positions of Kraepelin and Bleuler? My description of cases and my criticism of the positions of these authors presuppose a different way of looking at mental disease and the relation of psychologic alterations to body and brain, whether these alterations are temporary or permanent. The stimulus to our position was given by an American psychiatrist, Adolf Meyer, who began, in 1906, to formulate what he designated as a dynamic interpretation of mental disease. Meyer [14] said (1910) that unquestionably Kraepelin had delimited a group of cases that constituted a nosologic entity, which, while not so clearly delimited as Kraepelin had hoped, nevertheless, still offered a sufficient number of common characteristics to give a warning

13. Bleuler, E.: Zur Unterscheidung des Physiogenen und des Psychogenen bei der Schizophrenie, Allg. Ztschr. f. Psychiat. **84**:22, 1926.

14. Meyer, Adolf: The Dynamic Interpretation of Dementia Praecox, Am. J. Psychol. **21**:385 (July) 1910.

of the tendency to deterioration. He further pointed out that Kraepelin had not formulated the disease picture in terms that could be made into a scientific causal sequence, but that he had always to hide behind hypothetic toxins. Meyer said that, whereas evidence of toxins was not forthcoming, he found definite factors that were "apt to shape or undo a life—specific defects or disorders of balance, with special tendencies and habitual ways of bungling and substitutions, and a special makeup which is liable to break down in specific manners." He suggested that "complete action gets more and more disorganized by, first, trivial and harmless subterfuges or substitutions, which, in some individuals, lead further, to become harmful and then uncontrollable." At this same time, he referred to the catatonic reactions that were held to be so inexplicable psychologically, and pointed out that catatonia is not by any means far from a psychobiologic interpretation. It is in fact "closely related to what is seen in hypnotic states and in mystical fancies," and like "stages in religious symbolism and feelings of submission to influence by mystic power." Meyer, realizing that even the psychoanalysts turned to toxins and heredity to explain why serious complex material developed in special persons and then went on to a disastrous outcome in deterioration, emphasized the fact that psychologists and psychiatrists had failed to take into account the duration and intensity of psychobiologic facts and processes and had therefore turned to alterations of brain to explain the final outcome of fixity of process. He saw in habit conflicts and habit disorders, on the one hand, a set of factors "which preponderate in the side-tracking and curbing of leading interests, and the creation of disastrous substitutions"; and then, on the other hand, he recognized definite specific complexes in special cases as playing a "special dynamic rôle."

Meyer saw the human organism as a totality, integrated at successively higher and higher levels. For each level of organization or integration there are specific types of reaction, and the stimuli for each level are specific. Each new higher level works with the level below, but integrates it for more complicated and different purposes. Food and the reaction of the digestive apparatus play one part at the physiologic level, but a different one at the psychologic level. The latter makes use of the physiologic facts but has to consider additional factors that arise from the nature of the higher psychologic integrations. Definite lesions of the brain play the dominant rôle in certain types of mental reactions because they interfere with gross mechanisms without which the personality, integrated on the psychobiologic level, cannot operate. The damage reduces the capacity of the organism for the more complicated responses of the higher integrations. One cannot view art without eyes or ears, and yet neither the visual nor the auditory apparatus constitute art appreciation; memory is lost in certain widespread dam-

ages to the cerebral cortex, but the behavior of the organism depends on how this damage interferes with the general organization of the patient's whole life. For Meyer, the psychologic or psychobiologic facts of life—those facts which represent the responses of the total organism at this mental level—had as much force and validity as the facts organized at the level of physical and chemical or physiologic life. The laws of all of these levels are formulations of sequences more or less immutable. When the facts of one level seem to contradict the facts in another sphere, one does not discard them as invalid, but seeks through further knowledge to reconcile them. Biology does not renounce life because it cannot bring all facts into line with chemistry; neither does psychology give up any of the facts of its field because brain pathology denies their validity.

It is a common error of those who seek to find the explanation of psychobiologic facts in terms of body changes to assume that those who oppose them think that these facts are totally unrelated to neurologic and physiologic mechanisms. Nothing could be farther from the truth. I cannot conceive of any psychologic or neurologic reaction of the human organism that does not make use of specific neuromuscular pathways. The arousal of these reactions can take place only on certain definite stimuli, and they follow definite laws of order. This, however, is far from assuming that each reaction or set of reaction patterns is crystallized in the central nervous system. At the psychobiologic level, a special type of reactivity exists, which is called habit formation. Patterns can exist that are functionally fixed without being structurally set. This means that they can be modified and are modified, if one attacks the habit at the correct point and time.

The fact that the patterns that become fixed in the schizophrenic reactions are unmodifiable when they reach the psychiatrist is not an argument that they must necessarily have had a basis in structural alterations. Take, for example, the learning of language. That the learning of a specific language, German, English or French, is a psychologically conditioned process does not need proof. If one tries to modify this pattern after the fourteenth year of the person's life, it can never be done completely. One may speak a foreign language fluently, but one never drops traces of the accent of the mother tongue if one learns the foreign language after the twelfth or the fourteenth year. The language habit or pattern is therefore a psychobiologic habit that becomes functionally extremely rigid, but it is not exclusively structurally fixed. The interrelationship of the neurologically conditioned responses and the psychobiologically conditioned habits is well illustrated by this example. It is well known that in certain cases of damage of the brain involving gross destruction of tissue there is marked interference with the language function. An aphasic patient may lose

completely an acquired language and be able to speak only in the mother tongue of childhood. This gives evidence that a break in a psychobiologic function may occur at a lower level, but the fact that it breaks at a neurologic level does not preclude a break at a psychobiologic level.

The recent experience of those who have worked with habit formation in children, both normal and abnormal, has made it clear that habits that on the surface seem malignantly fixed in asocial and psychotic ways can be modified in an amazing fashion, if the habits are broken into radically and at a sufficiently early age. Dr. Esther Richards, working with ordinary children of the dispensary, has accomplished notable results in the treatment of asocial behavior and psychotic trends by a common sense correction of environmental factors and a substitution of normal, wholesome outlets and compensations.

It is clear that, in each case, one is working with a separate person with an environment that, in its complicated interplay of emotional forces, is specific for this person and different from that of other persons; that each person is being subjected to influences that are constantly making or breaking habits, intellectual, moral and emotional; that these habits are in a constant process of evolution and dissolution; that unhappy circumstances or forces may at any time drive this growing, changing person into channels for which he is inadequately trained or into alternatives for which he has no adequate organization or preparation. These circumstances may be found in a break in the organic or physiologic integrity of the body, or, more specifically, of the brain, or they may be found in human situations; but in each case it is a human being who reacts to them and he reacts as a sum total of all his constitution—i.e., as a psychobiologically integrated individual.

At the clinic, therefore, we describe reaction patterns or types and see in these reactions all the life factors, not only patterns that arise out of the set physical organic structure of the individual person, but also patterns that have a wide range of functional variability depending on the habits that have been formed on the foundation of the organic structure; we study the fixity of these habits and the circumstances that have produced them, and what circumstances can make for their modification.

The problem, then, becomes not whether the cerebral cortex is involved, but whether it is involved primarily. If it is, we make use of this fact and speak of reactions that are primarily organic, and in which psychobiologic factors are secondary as an expression of the neurogenic disorder. We set about reeducation, if sufficient mechanism remains, and we simplify environment to meet the new integrations of the personality.

We approach the problem of schizophrenia in exactly the same fashion. We use all the facts in the patient's life history and see him as a totality. We try to understand his abnormality in terms of known

trends of normal and abnormal behavior; stupor does not suddenly become the manifestation of a mystical toxin—it is like a hypnotic state; the schizophrenic thinking does not become an expression of acute or chronic cortical disorganization, but is like the thinking of dreams and primitive peoples; impulsiveness and autistic thinking are regarded as analogs of the undisciplined behavior and imagination of children and of childlike adults; persecutory systems become an exaggeration of normal human fears, and so forth.

I can now formulate the reaction tendencies that have been called schizophrenic. I purpose to substitute for this term, however, the term *parergastic,* suggested by Adolf Meyer in his last publication.[15] This term, using the Greek word ergasia for activity, suggests the dynamic elements of this psychobiology, and uses the prefix para—meaning "out of the way," or "paradoxical." The parergastic reaction is seen as a type of reaction arising from poorly developed habits of adjusting to other people and to new circumstances, with a tendency to avoid by retiring from, rather than meeting situations by decision; it is seen as arising further from an underdevelopment and undertraining of adequate affective responses to situations and people. It is characterized by an absence of moods, unless the situation is acute, and then by outbursts (rage, temper tantrums or sullenness); by a tendency to seek compensatory satisfaction in ruminating and daydreaming speculations; a tendency to avoid concrete activity; a willingness to live in an imaginary world without any translation of wishes and desires into realized ambition, and the development of a sexuality divorced from other people (autoerotic). This gives rise to an attitude of passivity—a rejection of forces that compel activity, unless these forces are so strong as to require active rejection in order that the inner poorly balanced imaginative life may be maintained; even then this activity serves only as an extruding process, to make the person more comfortable for his passive attitude. I therefore see the passivity component and attitude as the primary principle of the parergastic reaction.

As far as this principle operates to the exclusion of all others in a particular case, so far is the process malignant. If the past record of the patient has shown tendencies that make for more effective rapport with the environment and with people, this modifies the malignancy of the process; if the psychosis shows dominant affect and not just transitory, whimsical emotional response, this makes it a more benign type of reaction; if suddenly arising circumstances produce the break, telescoping life, as it were, one can count on readjustment to the extent that the

15. Meyer, Adolf: Genetisch-dynamische Psychologie versus Nosologie, Festschrift für Emil Kraepelin, Ztschr. f. d. ges. Neurol. u. Psychiat. **101**:406, 1926.

patient's reactions in the past have been adequate for the ordinary insults of life, and to the extent that the precipitating and alterable causes are unusual and real; if the break occurs gradually under the simplest environmental circumstances, one has little reason to hope for recovery, unless the patient is young enough to be completely retrained. The retraining of adult habits or character is difficult enough under the most favorable circumstances, and is almost impossible if the habits have developed distortions or dissolution over a period of years.

To rephrase this statement in psychiatric terms, one can say that affective admixtures argue for benignity; that an acute onset is prognostically better than an insidious onset; that a shut-in personality is more unfavorable than an outgoing personality; that, as far as the picture is ruled by passivity thinking—with a feeling of being forced and acted on by the outside world—so far is the development dangerous and likely to go on to deterioration of interest. Acting or behaving in a passive way, as if controlled from the outside, is as ominous, and one finds it accompanied by the development of subterfuges to avoid concrete contact, in the form of delusions or hallucinations, or a distorted hypochondriasis. These are extrusions of rejected, disturbing wishes and thoughts.

Referring again to the cases I have presented: Case 1 showed a slow, insidious development without any habits of work, interests or adequate affective rapport with people, and with the growth of unbridled and uncontrolled fancies and a growing tendency to the passivity attitude. Here one could not expect any recovery, for balancing factors were lacking.

Case 2, that of the English sailor, showed a more active type of personality, under the strain of a sex conflict and profound disillusionment, reacting with a sudden outburst of fantasy. The interest in working, the capacity for deep feeling and caring and the possibility of a favorable solution of the circumstances worked for readjustment, and at the end of a year the report of a recovery was received.

Case 3, that of the woman who communicated with her husband through the ouija board, then through automatic writing and finally through voices, showed a protracted dissatisfaction with a legitimate loneliness, a fair amount of working drive under necessity but not an independent working impulse when the necessity had passed (with her daughter's marriage), a tendency to exaggerate symptoms and to become inactive (sick) to meet lack of sympathy, and then the compensation in a pseudonormal way by communications through mystical channels. Here one could expect a partial readjustment because the way of departure was somewhat natural, the loneliness was legitimate and the contents of the fancies were not too far removed from the socially and personally acceptable fantasy of the patient.

Case 4, that of the engineer who wanted to be reborn, showed a consistent affect (depression), a really difficult situation in the approaching marriage and the record of proud ambition and of habits of work, with a drive to accomplish his objects. These circumstances were sufficient to indicate recovery. The sensitiveness, the overattachment for the mother and the tendency to withdraw from people and go in for rumination were factors which made this patient a candidate for further trouble if difficulties should arise, unless he could be retrained. This is difficult when the patient has lived with his bad habits for thirty-five years.

This, in broad outline, is a statement of three theories represented under the names dementia praecox, schizophrenia and the parergastic reaction. The first represents an attempt to explain a group of cases as a disease entity; it rejects psychobiologic facts as irrelevant and meaningless. The second admits the rôle of mental factors, but assigns to them a secondary place—the first place going to a hypothetic alteration of the brain or a toxin. The third view takes into account all the factors of a life, views them, as far as possible, as if they were a scientific experiment or causal sequence of events; gives due credence to any lesion of the brain or symptoms when a lesion of the brain exists, but utilizes the knowledge of more complicated psychobiologic integrations and reactions as factors which do exist and function, and which have scientific validity. It points the way to an experimental attitude and a rational therapy. It provides a place for organic etiology if this type of etiology can be discovered or demonstrated, but it refuses to accept a fatalistic attitude based on insufficient or nonexistent facts, with neglect of an etiology of a definite and valid sort. It reclaims for the realm of science human experience and the psychobiologic forces that go to influence it.

News and Comment

FIRST INTERNATIONAL CONGRESS ON MENTAL HYGIENE

The First International Congress on Mental Hygiene will be held in Washington, D. C., May 5 to May 10, 1930, inclusive.

Abstracts from Current Literature

EXTENSOR REFLEXES IN THE FORE-LIMB. D. E. DENNY-BROWN and E. G. T. LIDDELL, J. Physiol. **65**:305 (June) 1928.

In this paper the authors describe their further studies on the propensity toward extension in the fore limb of cats on stimulation of the ipsilateral nerves. Using decerebrate cat preparations, they isolated one muscle of the left fore limb, usually M. supraspinatus, M. biceps brachii or M. brachialis, and the nerves selected for stimulation. It was frequently found that when the median nerve was stimulated at the level of the wrist joint, i. e., after it had given off its branches to the forearm, it evoked a contraction in the ipsilateral supraspinatus, which might develop an active tension of as much as 0.75 Kg. within one second. The development of tension is gradual and may on occasion bear all the features of the process of "recruitment" of motor units in the crossed extensor response of the lower limb; that is, a long latent period, the sigmoid rise to a plateau, and a considerable after-discharge. Often, however, it is not so typical and on withdrawal of the stimulus there may be a marked rebound. The presence of a rebound after the stimulus shows that there has preexisted a state of inhibition. The dependence on a moderately high degree of initial tension for its appearance seems to be an essential characteristic of the reflex, and the degree of stretch is proportionately higher than that needed for any other reflex so far investigated by these workers, e. g., the crossed extensor response. This marked dependence on the degree of initial tension, which is itself in large part the expression of resting tone, suggested that modification in the response might be obtained through the afferents of the neck or of the labyrinth. Denny-Brown and Liddell, however, were unable to get constant results to verify this.

A characteristic of this reflex is the susceptibility to the concurrent stimulation of other ipsilateral nerves. At times it was possible to obtain with a weak stimulus a rise of excitation from the whole median, equivalent to, or greater than, that produced by its wrist division. This excitation could be totally inhibited by adding an intercurrent stimulus to the forearm branch of the same nerve. It was evident that the excitation of the whole trunk with a weak stimulus did not involve the fore limb branch, and they suggest that the varying degrees of excitation and inhibition resulting from stimulation of the whole median trunk are the result of conflict between the two factors found almost pure in its lower divisions.

Denny-Brown and Liddell next proceeded to investigate the part played by the median nerve at the ultimate distribution in the digits as digital nerves. If any one digital nerve was stimulated, the ipsilateral contraction of the supraspinatus was elicited. The digital nerve has a wide distribution centrally to the spinal motor units as shown by a notably high tension elicited by its stimulation. Ipsilateral excitation of the median field at the digital clefts and external aspect of the forearm will also excite the supraspinatus, confirming the excitation from the stimulation of the whole nerve trunk at the wrist. Also, a nociceptive mechanical stimulus (pinch) produces a brief response in the supraspinatus when applied to the forepaw, the nature of the response varying according to the position of the neck. The level of decerebration does not seem to bear a relation to these effects on the supraspinatus.

As the median nerve had this peculiar action in exciting a supposed extensor of the ipsilateral side, they explored its effect on a flexor muscle of the elbow, M. brachialis anticus. The ipsilateral median nerve, by its wrist division, seldom elicits any large excitatory response in this flexor in decerebrate preparations.

The authors then investigated the action of the ulnar nerve. In general, the ulnar nerve, stimulated anywhere in the fore limb, exerts an action more markedly inhibitory than the median at the wrist. Its powerful antagonism to the median is

even more striking when the digital nerves derived from the ulnar nerve are stimulated; such stimulation produces an inhibitory action on the spinal motor units of the supraspinatus, showing that the central connections of the ulnar nerve have an intimate relationship over a large part of that group of final motor neurons. Further exploration of the ulnar border of the fore limb showed that it had an inhibitory action on the supraspinatus more proximally in the area supplied by the internal cutaneous nerve. The responses are more closely similar to small ipsilateral contractions in the muscles of the hind limbs, where the nerves are more purely inhibitory.

The complete musculospiral nerve stimulated maximally in the arm is the most powerful inhibitor of the supraspinatus. Thus, the musculospiral nerve, inhibitory as a whole, and yet excitatory in its relatively small cutaneous division, has the same two contrasted properties as have the trunk and wrist division of the median nerve.

Myographically, they found, as a rule, that an ipsilateral nerve may have a considerable excitatory overlap with its neighbor in the motor units of a flexor, and only a small degree of inhibitory overlap. An ipsilateral nerve, which, as a general rule, is an inhibitor of the brachialis anticus, is the internal cutaneous nerve. Thus, an inhibitor of the shoulder's extension, it is an inhibitor of the elbow's flexor. It seems peculiar too in its proneness, at rather weak strengths of stimulation, to promote running movements in the hind limbs and occasionally in the fore limb. The musculospiral nerve, on account of its enormous homogeneous, mutually overlapping flexor exciting component, has, when excited maximally, a preponent extensor inhibition and flexor excitation.

Some experiments were done in furtherance of the observation that the biceps brachii has an unexpected influence; when stretched, it produces an inhibitory fall in its fellow muscle, the brachialis anticus. Denny-Brown and Liddell, who had considered that the biceps brachii is a typical flexor of the elbow, were surprised to find that the muscle showed many of the reflex activities which are associated with extensor muscles, especially the supraspinatus. For instance, stimulation of a nerve of the opposite fore limb produces types of tension development which are not uncommon with extensor muscles. Another feature which distinguishes the biceps brachii from most flexor muscles is the facility with which a stretch reflex can be elicited, this holding for slow stretch as well as for pluck or tap. They also obtained a number of records in which the rise of tension in the biceps in response to stimulation of the ipsilateral ulnar nerve is closely similar to the recruitment rise of the typical extensor, the vastocrureus. They believe, however, that their preliminary observations on the biceps brachii show that, of its reactions, a moiety is extensor-like rather than flexor-like.

These workers conclude that in the fore limb there can commonly be found departures from the rule which obtain largely in the hind limb, that electrical stimulation of a nerve of the limb promotes withdrawal (flexion) on the same side. The median nerve at the wrist, stimulated tetanically, evokes a reflex contraction in an ipsilateral extensor (the supraspinatus) of considerable tension, the tension being of gradual onset. The forearm branch of the median nerve inhibits the reflex, as does stretching of the flexor digitorum profundus. The branches of the median nerve in the digital clefts excite the reflex to a high degree, while the ulnar nerve and its branches in the digital clefts inhibit it. The brachialis anticus in its response to nerve stimulation behaves as a flexor muscle, while the biceps brachii is not a typical flexor as it shows a marked response to the stimulation of a nerve of the opposite forelimb as well as a maintained stretch reflex.

ALPERS, Philadelphia.

CONCUSSION OF THE BRAIN, OR "PUNCH DRUNK." Editorial, J. A. M. A. **92**: 314 (Jan. 26) 1929.

Concussion of the brain or spinal cord has always been shrouded with considerable mystery because the mechanism of its production has not been understood. For years physicians have believed that concussion may be solely a functional

change without any proper basis in structural alteration. Founded on such ideas, fraudulent claims for alleged disability following concussion of the central nervous system have enjoyed a long and amazing prosperity. Consequently it is not surprising that the wide diffusion of reliable information about this subject accomplished by the recent publication in *The Journal of the American Medical Association* of the article by Martland (Martland, H. S.: Punch Drunk, J. A. M. A. **91**:1103 [Oct. 13] 1928) has resulted in much favorable comment. As Martland acknowledges, pathologists engaged in medicolegal work have long been familiar with the perivascular intracerebral hemorrhages that he has illustrated and described. To them concussion of the brain has been simply another way of saying contusion or bruising of the brain. They have been accustomed to finding widely disseminated minute hemorrhages as the only change in casual routine examinations of the brains of persons who die in coma from injuries of the head which have left the cranial bones unbroken. They have also learned that demonstration of the hemorrhages in some of the brains is best attained by microscopic examination. Credit is given by Martland to Cassasa (Cassasa, C. B.: Multiple Traumatic Cerebral Hemorrhages, Proc. New York Path. Soc. **24**:101 [Jan.-May] 1924) for his description of similar lesions. Martland found these small hemorrhages in the brains of nine of 309 persons who died from injuries of the head. They were in or near the corpora striata, rarely or never in the cerebral cortex and never in the cerebellum and brain stem, which are covered over and held so snugly by the tentorium cerebelli. There were no broken cranial bones about any of these nine brains.

The association of these minute bruises with the sequences known for a long time as "punch drunk," a colloquialism among those occupied in one way or another with prize fighting and professional boxing, was a manifestation of discriminating judgment. Martland's suggestion of this relationship was inspired in part by the reasons that Gene Tunney, according to the lay press, gave for relinquishing the championship: his own experience with concussion, an amnesia of two days, and the fear that continued blows on the head would eventually unbalance his mentality. Thus we are continually reminded of the way in which progress is made unexpectedly by a synthesis of observations in fields that apparently are wholly unrelated. This relevancy of the gossip of the prize ring to the knowledge of concussion possessed by a few pathologists specializing in medicolegal work, knowledge that has heretofore found but little clinical application, may have surprisingly rich consequences. One outcome is likely to be a clear portrayal of one more danger to which boxers are subjected in addition to those which have been described repeatedly in medical literature (Wolff, K.: Todesfälle durch Boxkampf, Deutsche Ztschr. f. d. ges. gerichtl. Med. **12**:392, 1928). This will result, no doubt, from a thorough study, which all the modern highly technical methods now practiced, of the brain of some prize fighter afflicted with "punch drunk."

The experiments have been and are still being made, and the animal this time is the genus *Homo*. It will be recalled that tapping the head of lower animals with a hammer at frequent intervals and with violence insufficient to bring about serious bruising will cause unconsciousness. Martland suggests, as have others also, that forces operating under the laws of hydrostatics cause the hemorrhages of severe concussion. But waves in the fluids within the cranium due to force applied to the outside of the head may not attain size and vigor sufficient to tear the blood vessels. Such waves in the blood confined in vessels, especially large veins, may interfere with the normal flow only enough to produce a transient interference with the nutrition or oxygen supply and slight dizziness or momentary interruption of consciousness. Waves in the intraventricular fluid or in that of the leptomeningeal cisternae may have their own particular consequences anatomically and clinically. Another factor, which Martland fails to mention, is the difference in weight between the gray and the white matter. This difference, although not great, probably explains the location of hemorrhages where the gray and the white matter join (Apfelbach, C. W.: Studies in Traumatic Fracture of the Cranial Bones,

Arch. Surg. **4**:434 [March] 1922). Since they possess a different inertia, the degree to which they are jolted out of their normal positions by blows on the head also varies.

The changes in the brain and its membranes from trauma are profoundly influenced also by the rigid manner in which the falx cerebri and the cerebral blood vessels and cranial nerves are fastened to or within the cranial bones. Even slight displacement of the brain causes distortion of the channels of blood vessels and modifies the current in them where they emerge from bony channels or dural sheaths. Changes of the cranial axes by external violence also contribute to such disturbances. The large subdural hemorrhages that compress one or both sides of the brain, erroneously called for many generations hemorrhagic pachymeningitis, are now generally recognized as traumatic. They are probably due to tears in the cerebral veins where they pass across from the tightly bound superior longitudinal sinus to their bed in the leptomeninges. For a short distance between these two places they are quite devoid of support. The pull on nerves firmly placed in their paths in the pons below the tentorium cerebelli must be transmitted to their more proximal intracerebral portions when violence piles up to the cerebrum a little more in some than in other parts of the cranium. The completeness with which the brain is surrounded, the fact that its envelop is in part firm bone, distention of the cervical and cerebral veins or its converse, the development and character of the muscular and other attachments of the head to the trunk, and the direction of the violence are some of many conditions by which cerebral concussion is influenced. The resistance of blood and cerebrospinal fluid to compression is also highly important. Consequently, the mechanics of concussion is involved. With due consideration of the structures and forces implicated, considerable appreciation is possible of what takes place, as well as the manner of concussion and the reasons for it. But a new era in our knowledge of this subject is bound to begin with accurate investigation of the brains thus affected, of the early and late changes, and especially of their topographic distribution. To these there should be added reliable details about the nature of the violence concerned in producing the pathologic changes in the tissues.

CHAMBERS, Syracuse, N. Y.

MORE REFLEX EFFECTS OF ACTIVE MUSCULAR CONTRACTION. SYBIL COOPER and R. S. CREED, J. Physiol. **64**: 199 (Dec.) 1927.

In an earlier paper the authors showed that proprioceptive impulses, set up by the active contraction of a cat's hamstring muscles, readily inhibit decerebrate tonus in the quadriceps extensor muscle of the same side. In this work they repeated and extended the experiments, using a high frequency isometric myograph with an optical recording system for more delicate means of recording.

The hamstrings of decerebrate cats were made to contract by faradic stimulation of the seventh lumbar or first sacral ventral roots, and the ensuing changes in the tension of the patellar tendon were recorded. They found that the active contraction of the hamstrings reflexly inhibits tonus in the vastocrureus, and this effect is more marked when the former muscles are prevented from shortening. In the isometric record of this inhibition there is frequently an initial plunge of a steepness much greater than that of the later course of the decline, and the ensuing "inhibitory recruitment" may be gradual and prolonged. The recovery, like the initial fall, often occurs in a series of steps. Sometimes there is no recovery, and the muscle is left in an atonic condition. Very rarely, a marked rebound contraction brings the tension to considerably more than its previous resting level. This occurrence of recovery and an occasional rebound is interesting, because the inhibition of the myotatic contraction of the quadriceps by the passive stretch of a knee flexor was found by Liddell and Sherrington to be followed by almost no recovery. For the elicitation of the reflex in an active preparation, the stimulus must be somewhat stronger than is required to produce a minimal contraction of the hamstrings. The mechanical latency tends to become shorter as the intensity of stimulation is increased. The surprising feature of these myograms is the final decline in tension

on withdrawal of the stimulus. Cooper and Creed believe that this final fall in tension is a true physiologic event and not a mechanical artefact. They do not believe that it is an expression of simultaneous excitation and inhibition in which the latter outlasts the former, but can offer no other explanation for it at present.

In a further series of experiments they found that the contraction of the vastocrureus in a crossed extensor reflex (decerebrate cat) is also inhibited by hamstring contraction. Experiments performed to see the effect of the vastocrureus on the semitendinosus failed to show any evidence that an intercurrent contraction of the vastocrureus modifies the course of the flexion reflex.

When a motor root supplying the hamstring muscles in a specially prepared specimen was stimulated, contraction of the hamstrings was followed by a well marked reflex contraction in the sartorius. The reflex effects of hamstring contraction were shown to be greater when these muscles were prevented from shortening. This is also true of the effect on the sartorius. Contraction of the sartorius was observed on a mere passive stretch of the hamstrings. Magnus states that in the fore limb of an intact animal or in that in an animal in which the thalamus has been removed, the passive stretch of one digit flexor causes a reflex contraction of the whole group of flexor muscles in the forearm.

On causing the tibialis anticus to contract by stimulation of the seventh lumbar root, the sartorius responded by a contraction. The mechanical latency was always notably longer than that of the similar response to stimulation of an ipsilateral afferent nerve or sensory root. If the tibialis anticus was permitted to shorten (after detachment) the contraction of the sartorius was generally smaller. Cooper and Creed found that the passive stretch of the tibialis anticus was without effect on the sartorius. If all other muscles of the limb have been satisfactorily denervated, section of the peroneal nerve abolishes the reflex.

On making the left vastocrureus contract by stimulation of the peripheral stump of the cut sixth lumbar root, they were able to get records of well marked relaxation of the right vastocrureus, sometimes followed, on withdrawal of the stimulus, by a rebound contraction. The authors recall the fact that the proprioceptive fibers from extensor muscles are of two kinds. One set carries impulses which reflexly induce shortening of the very muscle in which they originate (tonus, "shortening reflex") and at the same time cause reflex relaxation, or more rarely contraction, of the fellow muscle of the opposite limb. The other set carries impulses which reflexly inhibit the muscle in which they originate. In the experiments here reported it is evidently the endings of the former set that were stimulated by active contraction of the vastocrureus muscle. In reflex stepping, therefore, the act of extension in one limb will initiate a volley of centripetal impulses tending reflexly to facilitate simultaneous flexion of the opposite limb. During the flexion phase possibly the passive stretch of the knee extensor may evoke Philippson's reflex and so assist in the simultaneous extension of the opposite limb.

By stimulation of the left hamstrings, Cooper and Creed were able to observe a definite relaxation of the right tonic extensor muscles. Sherrington has reported inhibition of the knee reflex as a result of the stimulation of the afferent fibers in the contralateral hamstring nerve. On the other hand, he and Liddell were unable to detect any influence, either inhibitory or excitatory, of a passive stretch of the knee flexors on the contralateral stretch reflex of the knee extensor.

The occurrence of a sartorial "jerk" in spinal cats was reported and illustrated.

ALPERS, Philadelphia.

NUTRITION AND LONGEVITY. Editorial, J. A. M. A. **92**:57 (Jan. 5) 1929.

Heredity, external environment and nutrition are significant factors in determining the length of life. The significance of heredity is emphasized when the mode of inquiry is that of gathering data relating to persons who, at the time of selection, have already attained to noteworthy longevity. Without necessarily questioning the conclusion thus reached that heredity is probably the dominant factor in

longevity, the thoughtful student will not fail to reflect that this method of investigation is fitted primarily to bring to light any correlation which may exist between the longevity of the individual and that of his parents and grandparents, whose ages at death will usually stand out as clear-cut quantitative data in such an inquiry. The method is less able apparently to establish correlations with the factors of external environment and of nutrition or food habit, because the environmental conditions of a lifetime cannot, in normal human experience, be ascertained with anything approaching the degree of definiteness with which hereditary factors may be determined. Hence, the fact that inquiries of this sort heretofore have failed to establish satisfactory correlations between environment and longevity does not prove that the relationship does not exist; it is at least equally probable that the environmental factors may actually have a greater influence than has been demonstrated. Unfortunately, not all discussions of longevity have shown this discrimination. Too often the type of inquiry referred to has been taken to justify the view that nothing which a person can do for himself, or which can be done for him during his lifetime, will materially influence his length of life—a conclusion that far overreaches the available scientific evidence and carries a flavor of dogmatic fatalism rather than of modern medicine. Much of the available evidence offers good reason for believing in environment as a factor in longevity. To mention but one of many causes of death, is there not full justification for believing that many men who have died of pneumonia might have lived longer if they had been less exposed to infections and bad weather?

Broadly regarded, the nutritive condition of any person may be (doubtless in many cases is) influenced by inherited constitution and by external environment, as well as by the nutriment which he consumes. Moreover, even within the range of variation of what is considered a normal and adequate food supply, it now seems to have been demonstrated that the food which one consumes may influence one's longevity. Lifelong experiments and observations have not been made on human beings, for with them it is not feasible either to control the food intake for a lifetime or to find cases in which all other conditions have been constant while food habits have differed in known and definite ways. Both these conditions can be realized, however, in laboratory experiments with rats, and the chemistry and physiology of human and rat nutrition are sufficiently similar (allowing for the fact that the rat lives its life in about one thirtieth of the time of man) so that a nutritional principle established by experimentation with one of these species may, with a satisfactory degree of probability, be expected to apply to the other species as well.

In a recent study (Sherman and Campbell: *Proc. National Acad. Sc.* **14**:852, 1928), about 400 rats, all of the same heredity, and living under conditions alike in all other respects, were kept in about equal numbers on two different dietaries—both nutritionally adequate wheat-and-milk mixtures but differing in the proportions of milk in the food mixture. Although the diet with the lower proportion of milk was definitely shown to be adequate, the animals receiving food containing the higher proportion of milk lived, on the average, 10 per cent longer—a significant gain in longevity. Critical examination of the results from a statistical point of view indicated that there was not more than one chance in a hundred that this result could have been accidental or due to the natural physiologic variability of the animals. The superior longevity induced by the improvement of the already adequate diet was further shown in the fact that much larger percentages, both of males and of females, attained definite standards of longevity on the improved than on the original dietary. The investigators also point out that this gain in longevity resulted from a single and simple dietary improvement, and that it is altogether probable that the better of the two diets here studied is susceptible of being still further improved. Hence, it is entirely reasonable to hope that further investigation may show possibilities of even more gratifying improvements in longevity, through the application of the constantly growing knowledge of nutrition.

CHAMBERS, Syracuse, N. Y.

THE ACTION OF LIGHT ON THE EYE: PART II. THE PROCESSES INVOLVED IN RETINAL EXCITATION. E. D. ADRIAN and R. MATTHEWS, J. Physiol. **64**:279 (Dec.) 1927.

In this paper the authors study the effect of flashes of light of short duration on the retina. The eye of the conger eel was used for the experiments. The eyes were normally kept in the dark, and intervals of a minute or more were allowed between successive exposures. The measurement they observed was that of the "reaction time" of the optic nerve, i. e., the period between the beginning of the exposure to light and the beginning of the nerve or retinal response.

When the exposure is of long duration (one second or more) a reduction in the intensity of the light lengthens the reaction time. If they used an illumination giving a fairly short reaction time and tried the effect of reducing the duration of the exposure, the reaction time remained unchanged as long as the duration of exposure exceeded a certain critical value determined by the intensity of the light. With exposures shorter than the critical duration, the reaction time was lengthened. For a given intensity there is a critical duration which the flash must possess if it is to have the maximum stimulating effect judged by both the shortness of the reaction time and the frequency of the impulse.

In measuring the retinal reaction time, Adrian and Matthews found that the nerve discharge is preceded by the development of the initial negative electric response of the retina, and that the retinal-nerve interval remains approximately constant for all intensities of light, i. e., for all values of the reaction time. This is true of exposure of long duration and for flash also. It is found that a given reaction time implies a given magnitude of discharge, whether this is caused by a bright flash of short duration or a dim flash of longer duration.

They conclude that, for short exposures, the stimulating effect, judged by the discharge of the optic nerve, depends simply on the total quantity of light received by the eye; this result agrees with the relation found in man between the quantity of light received in a flash and the brightness of the resulting sensation.

From this point they considered two processes: a primary change coinciding with the exposure — the light product, or light effect — and a slower, secondary change following on the primary and ultimately producing the response. The authors refer to the work of Hecht on two invertebrates and quote his conclusions that the primary change is a photochemical decomposition and the secondary change is a distinct chemical reaction. The results of Adrian and Matthews point to primary change of the same kind, except when they used flashes as long as the critical duration. In the secondary change, too, they agree with Hecht's hypothesis, for in flashes of short duration there is a linear relation between the reciprocal of the retinal reaction time and the quantity of light in the flash. This means that within certain limits the velocity of the secondary change is a linear function of the amount of light product formed. Since the duration of the process is so simply related to the quantity of light, the whole of the time is probably taken up by a single reaction.

In studying the relation between area and intensity, and using continuous illumination, Adrian and Matthews found that with retinal images up to 1 mm. in diameter, an increase in size had the same effect as an increase in the intensity of light — the maximum frequency of the impulses increased, but not in direct proportion to the area, and the reaction time became shorter. Using brief flashes they found that, for a flash which is shorter than the critical duration, a given quantity of light produces the same reaction time whether it is presented at a high intensity for a short time, or at a lower intensity for a longer time. The relation between area and intensity can only mean that the total effect of the light is transmitted to some region the extent of which is independent of the area illuminated. The effect might be diffused widely over the retina or concentrated into a small number of nerve paths which have an overlapping distribution on the retinal surface. In either case, the transmission of the light effect must take place rapidly as part of the primary change, and the secondary change must occur in the region to which the light is transmitted.

ALPERS, Philadelphia.

EXPERIMENTS ON HALLUCINATIONS. K. ZUCKER, Arch. f. Psychiat. **83**:706 (May) 1928.

The author approaches the problem with the idea that hallucinatory experiences can be investigated experimentally on an objective basis, and that such an investigation could show differences between hallucinations in different diseases which cannot be obtained by merely questioning the persons experiencing them. The point especially investigated by the author concerns the reality value of the hallucinations. In some cases the hallucinations are described by the patients themselves as being something different from real objective occurrences. In others, however, the patients insist that the hallucinations are exactly like their objective perceptions. The question as to whether this is actually so served as the basis of the author's investigation. He selected carefully patients who repeatedly stated that their hallucinatory experiences were exactly like objective experiences. The content of the hallucination of the patient in question was then acted out in reality as perfectly as possible. The person who performed the imitation was not seen by the patient, who was observed during the process of imitation to see whether there was any change in his mode of reaction. After the experiment he was asked to compare his hallucinatory experience with the one caused by the imitation, stressing especially the reality value to the patient himself. Twelve such cases were selected, four of schizophrenia, one of "propfhebephrenia" (schizophrenic psychosis engrafted on a mental defect), one of paraphrenia fantastica, one paranoid involutional psychosis, one of senile dementia with hypochondriacal delusions, one of paresis, two of alcoholic delirium and one of delirium in a patient with frontal tumor.

In the schizophrenic patients the results were invariably the same: the patients showed by their facial expression and behavior during the experiment, as well as by their statements following the experiment, that there was a difference between the actual occurrence and the hallucinatory experience. In one case, for instance, in which the patient complained that electric currents were going through his body and that these currents came from wires placed in the bed, a procedure designed to imitate this hallucination was at once recognized by the patient as being "different." The patient had no way of discovering that somebody had actually tampered with his surroundings, yet he was able to distinguish by mere perception the difference between the actual and the hallucinatory experience. A similar result was obtained in the case of "propfhebephrenia," in the patient with paraphrenia fantastica and in the one with paranoid involutional psychosis. A diametrically opposite result was obtained in the case of alcoholic delirium. Here the hallucinatory experience apparently had the same reality value as actual experiences, and the patient could not differentiate the one from the other. In one case, for instance, in which the patient was imagining that he saw rabbits in his room, a real rabbit introduced into the room was judged as belonging to the others; both from the expression of the patient and from his statements one could not discover any difference between the effect produced by the real rabbit and that produced by the imaginary ones. Results more or less similar to those in schizophrenia were also obtained in one case of paresis with hallucinations and in another of senile dementia with paranoid delusions. Here, too, there was definite differentiation between the real and the imaginary perceptions; only a partial differentiation was possible in the case of delirium with tumor of the frontal lobe.

An interesting discussion follows the recording of the results, and the author concludes by stating that hallucinatory phenomena can be investigated by objective experimental methods, and that not only the reality value but also the actual occurrence of hallucinations (drugs, such as mescaline, etc.) as well as their relation to definite syndromes should be further investigated.

MALAMUD, Foxborough, Mass.

THE TREATMENT OF HEADACHE IN CHILDREN. R. C. LIGHTWOOD, Lancet **1**:770 (April 14) 1928.

Headaches in the young, especially in infants, are comparatively uncommon. Cephalic pain, which may be taken to include headache and earache, causes young

infants to put their hands to their heads, scratching them and rubbing them at times; it also causes wrinkling of the forehead, crying and restlessness. Earache is by far the commonest cause of screaming and of putting the hands up to the head; it is especially frequent in association with teething and catarrhs of the first two years. Apart from earache, true headache may occur in infants and is often a sign of an organic disease of the brain, such as meningitis, intracranial tumor or hydrocephalus. The persistent crying of syphilitic infants has been ascribed to headache resulting from meningo-encephalitis. Eye strain is the commonest single cause of headaches which are prolonged over more than a few days. It seldom occurs before the age of 5 years. The headache of ametropia is commonest after school hours, thus contrasting with toxic headaches which occur mostly in the morning.

A large number of conditions may be responsible for toxic headaches. All the acute infections may produce them, but less commonly than in adults. Fever of less than 104 F. rarely causes this symptom and if it should do so, suspicion of tuberculous meningitis should be aroused. Ordinary toxic headaches associated with an acute infection may be safely relieved by acetylsalicylic acid in doses of 5 grains (0.324 Gm.) every four hours in a child of from 5 to 10 years of age. A morning headache occurs in some children who have a prolonged disturbance of digestion. Such children are most commonly below the age of 7 years and are found to have furred tongues, foul breath and impaired appetite; constipation and abdominal distention may also be present. The digestive disturbance may be dietetic; in this case, proper food should be selected, and nothing should be eaten for two hours before bedtime. Stomachic mixtures are useful but subsidiary. Digestive disturbances may also be due to infections of the upper respiratory tract. They are common with chronically infected tonsils and adenoids, which result in the swallowing of secretions.

Headaches due to juvenile rheumatism occur in children of the hospital class above the age of 6 years, and usually in the early part of the day. For the headache of chorea, 5 or 10 grains (0.324 to 0.69 Gm.) of acetylsalicylic acid according to age, is useful. Migraine, while uncommon, is seen occasionally during the latter half of this period. It has a tendency to be accompanied by pyrexia. There is considerable evidence in support of the view that migraine is manifested in the child by cyclic vomiting. For recurrent bilious attacks accompanied by headache and due to acetonuria, dextrose is an efficient remedy. For young children, a teaspoonful of commercial dextrose three times a day should be used. Persistent headache is found in a certain small group of children with obesity; it is also a feature of dystrophia adiposogenitalis and is often complained of by girls at the age when menstruation commences. Endocrine therapy, analgesics and bromides may all be used in treatment. Headache occurs in about 50 per cent of all cases requiring operation for adenoids. Septic tonsils, dental caries, uremia, nephritis, anemia, trauma, lead poisoning, juvenile paresis and hysteria may all produce this symptom. Sinusitis is an exceptional cause in childhood and only occurs at the end of this period. The cause frequently is not discoverable. Occasionally a family history can be obtained.

PETERSEN, Montreal.

AMBLYOPIA RECIDIVE AFTER OPERATIVE REMOVAL OF THE OVARIES. A. ROSENSTEIN, Ztschr. f. d. ges. Neurol. u. Psychiat. **115**:13 (July) 1928.

Sudden transitory blindness in one or both eyes is not uncommon, and occurs without definite relation to constitutional type, age or race. In many cases it is due to a chronic progressive endarteritis of the central artery of the retina, and less often to syphilis. Sometimes it is due to emboli in branches of this artery, with recannulization of the emboli. It can be caused by aneurysm of the internal carotid. Rosenstein says that next to syphilis, multiple sclerosis is the most common of the infectious diseases to cause this condition. Rarely, malaria causes transitory blindness by vascular spasm. Variations in cerebral pressure, as by direct compression of the chiasm through a sudden pressure increase in the third

ventricle, can cause an amaurosis lasting from a few to several days. Choked disk, of course, is a cause of this condition. Among the toxic conditions, nicotine is an occasional and quinine a rare cause of amaurosis. Uremia, through retinal involvement and papillitis, causes toxic disturbances of this nature. Amauroses in intestinal intoxications have been reported. Migraine of the ophthalmic type, and of the local type by spasm of the calcarine vessels, causes transitory blindness.

A number of cases of transitory blindness have been reported in relation to menstrual disturbances. Rosenstein points out, however, that a coincidental occurrence does not mean a relationship between the two conditions. For most women, the menstrual period is one of decreased resistance to all infections. These eye disturbances during menstrual disorders have been looked on by some investigators as vicarious menstruation. This term must, however, be reserved for those cases in which there is regularly a true hemorrhage into or from another organ at the time at which the menses are due. In 220 cases from 1876 to 1926, Schroeder found vicarious menstruation in the eye in eight or 1.3 per cent of cases.

In spite of the great frequency of female genital disorders, periodic blindness has not been reported up to the present time. Rosenstein reports a case of a woman, aged 29, who in 1926 first noted migrainous attacks with dimness of vision which occurred very irregularly. About three months later she had a total extirpation of the uterus leaving an ovarian rest. One month after this she had blindness with her migrainous attacks, the blindness lasting from three to four days, and thereafter she had this periodic migraine and blindness every four weeks at the time of her menses. The visual disturbances begin and end with concentric shrinking of the visual field, and with loss of color vision, only the green remaining intact. The pupils become wide and almost stiff to light, while the fundi do not show any changes in either the vessels or the nerve heads. The convergence reaction of the pupils is decreased, accommodation is not interfered with, and the external ocular muscles are not involved in the attacks. Examination of the rest of the nervous system gave negative results, and the spinal fluid was entirely normal. Rosenstein attributes the attacks to a disorder caused by ovarian deficiency, and says that following ovarian therapy they were improved. He attributes the transient blindness to a nerve rather than a retinal disturbance and says that concentric narrowing of the visual field, as seen in this case, is characteristic of many diseases of the optic nerve, as for example in optic atrophy.

ALPERS, Philadelphia.

FRACTURE OF THE OPTIC CANAL. OTTO BARKAN and HANS BARKAN, Am. J. Ophth. **11**:767 (Oct.) 1928.

Fracture of the optic canal, with a resultant lesion of the optic nerve, would seem to be much more common than is generally supposed. Reduction in vision following injury to the head is usually due to a fracture of the bony canal with consequent hemorrhage into the sheath, to actual laceration of the nerve within the canal, or to both. In some cases, however, pressure of an anterior clinoid process may be the cause. Others have suggested a contrecoup contusion of the nerve, by its having been forcibly driven against the bony boundaries of the foramen, in those cases showing temporal or nasal blindness.

The history of a typical case is usually that of a blunt injury in the region of the orbit which may appear slight. It is also fairly frequent in fracture of the base of the skull, especially when due to gunshot injuries. "Forensically, injuries to the supra-orbital region are therefore very important because the injury may seem slight in comparison with the succeeding loss of vision. The patient, if he is conscious, notices immediate loss of vision either on regaining consciousness or after the swelling of the lids has subsided. Either the immediate almost complete loss of vision rapidly improves, in which case the cause must have been pressure of blood within the sheath with consequent functional inhibition of the visual impulses, or after the initial improvement of vision a permanent defect in

the field remains, in which case there must have been a tear in the nerve in addition to the pressure of the blood; or finally amaurosis remains as the result of complete severance of the nerve."

Ophthalmoscopically, a slight congestion of the disk may be found, but it has never been observed by the authors. Pallor due to descending atrophy may be observed from the second week on. A sector defect of the visual field at the beginning may be interpreted as due to a tear in the nerve and therefore as more or less permanent. Central scotomas may more rarely be observed. The authors conclude that a case showing a sector defect extending to and including the macular region is sufficiently characteristic to be almost pathognomonic.

The reaction of the pupil to light is dependent, it would seem, on the integrity of the macular bundle and affords evidence that the pupillomotor fibers are contained within this macular bundle.

The authors have been able to find reports of only about eighty cases in the literature; yet in the past six years they were able to diagnose this condition in twenty-two cases. Five illustrative cases are reported in this paper. They conclude that if careful perimetric fields were taken in all cases of fracture at the base of the skull, one would find a fair percentage of partial constrictions of the visual field. The treatment recommended is that of early exploration of the optic nerve and bony orbit, with evacuation of any blood found in the sheath of the nerve, and observation of the anterior clinoid process for a possible fracture, which might be present and yet not be demonstrated by the x-rays.

FERGUSON, Philadelphia.

SOME TENDENCIES IN NEUROPHYSIOLOGY AND THEIR RELATION TO NEUROPSYCHIATRY. WILLIAM MALAMUD and DAVID ROTHSCHILD, J. Nerv. & Ment. Dis. **68**:231 (Sept.) 1928.

In neurophysiology two tendencies are observed: one a special study of the function and dysfunction of a separate part of the organism, and the other the study of the function and dysfunction of the whole. Neurophysiology developed from the balancing action of these opposite yet not conflicting tendencies. On the one hand, there is an attempt to express phenomena in terms of elements and mathematical precision, and on the other, an attempt to appreciate physiologic phenomena as parts of a whole. With these conceptions, the authors discussed the general trends underlying the work of two laboratories.

1. Studies of the function of the special senses at the Heidelberg Neurologic Clinic: In this clinic, such investigators as Weizsacker, refuse to regard as a serious obstacle to investigation the impossibility of mathematical precision in such studies as those of the special senses. They discovered that in certain cases in which spatial perception is impaired a systematic reconstruction takes place in the patient's whole appreciation of space. Subdivision of the nervous system into its functional parts hence becomes inadequate and arbitrary. Sensorium and motorium appear interdependent. Pathologic disorders are seen to lead to new situations requiring the adaptation of a new organism as a whole. Disorders of function, therefore, come to be regarded more in the light of metamorphosis of function, than the mere subtraction of a part of function.

2. Neurophysiologic investigations at Frankfort-am-Main: At the Institute for Investigating Consequences of Injuries to the Brain, Goldstein and Gelb have undertaken an elaborate psychologic and neurologic examination. The fundamental principle emphasized is that for every local change there occurs simultaneously an alteration in the entire organism. Goldstein's conception of the nervous symptoms is that of a ganglionic network capable of all reactions without the necessity for one definite structure to find them. Thus, much variation in the patellar reflex can be found in healthy persons by changing the posture of the limb or modifying the attention of the subject. Goldstein prefers to regard these variations not as due to higher inhibiting centers but as definite reactions adapted to altered situations. In this light, the so-called tonic reflexes of Magnus and DeKleijn are by no means fixed, established reactions, but are dependent on the total situation.

Similarly, sense perception can be shown to be involved in cerebellar ataxia, and eye movements shown to cause changes in tonus in the arms. Aphasia, from this point of view, can no longer be regarded as a disturbance in any special faculty, but as the result of an alteration in the organism as a whole. Thus the work of these two German schools indicates how a change in point of view in the biologic sciences in general has affected the study of clinical neuropsychiatry.

HART, Greenwich, Conn.

THE SUBLIMINAL FRINGE IN SPINAL FLEXION. D. E. DENNY-BROWN and C. S. SHERRINGTON, J. Physiol. **66**:175 (Oct.) 1928.

In this investigation the authors sought evidence that the fringe of subliminal influence, which is traceable as a feature under weaker stimulation of the afferent nerve, has its counterpart also under maximal stimulation of the afferent nerve. Under conditions and with technic similar to those previously used, further experiments disclosed that a fringe of subliminal effect centrally accompanied the reflex discharge evoked just as much by a strong as by a weak stimulation of the afferent nerve. The internal saphenous and peroneal nerves were used for two experiments; the external plantar afferent and external cutaneous nerves for a third, and the posterior tibial afferent and musculocutaneous nerves for a fourth.

The observations showed that certain afferent nerves, studied in relation to the flexor motoneuron aggregate of the limb, have a potential field of excitation wider than the field of motoneurons which they actually discharge. These and other observations signify that the reflex discharge is determined by a grading of the central stimulation affecting the individual motoneurons of the motor unit aggregate. A factor in the grading for each motor unit would be the richness with which the terminals of the excited afferent nerve converge on that particular unit. A threshold stimulus to the afferent nerve causes, therefore, a motor discharge in those motor units on which the afferent terminals derived from the few individual low-threshold nerve fibers in the afferent nerve converge and overlap in richest number, that is, in those motor units which receive the excited terminals in greatest density. The subjection of the same nerve to a stronger stimulus brings into play additional afferent nerve fibers and therefore additional excited central terminals, some of them affecting the same central units previously excited and others the central units in which the convergence had previously been too sparse to bring about their actual discharge, that is, instances in which the excitatory influence was previously subliminal. The observations now adduced show that the maximal stimulation of the afferent nerve, the excitation of all the afferent fibers of the nerve, results not only in the discharge or supraliminal stimulation of the maximal number of motoneurons for which that particular afferent is then competent, but in an accessory fringe of subliminal stimulation of other motoneurons. The latter group is demonstrable in various ways, for example, by interaction yielding summation and facilitation. Perhaps it corresponds to the field of enhancement by which strychnine augments the fractional group.

In central stimulation of the motor units there are, therefore, three grades of stimulation: (1) maximal, which is abundantly above the threshold, (2) just supraliminal and (3) just subliminal.

ALPERS, Philadelphia.

CEREBROSPINAL FLUID IN INFANTS AND IN CHILDREN. A. LEVINSON, Am. J. Dis. Child. **36**:799 (Oct.) 1928.

The technic of lumbar puncture in children is the same as in adults, with the exception that a shorter needle is desirable and the puncture is made at a higher intervertebral space, preferably between the first and second lumbar vertebrae. In addition, cerebrospinal fluid may be removed by ventricular puncture in infants when the anterior fontanel is still open. Cisternal puncture is useful in some instances. Opinions vary as to the normal color of the cerebrospinal fluid in new-born infants and children. The author believes that it is

colorless and that a yellow color indicates some degree of cerebral hemorrhage. In older infants and children, the cerebrospinal fluid is the same as in adults.

In cerebral hemorrhage of the new-born infant, the cerebrospinal fluid varies from a moderate degree of xanthochromia to bloody. In hydrocephalus the cerebrospinal fluid is normal except for an increased amount. In amaurotic family idiocy and in mongolian idiocy the author found the fluids normal. In spasmophilia there is a doubtful reduction of calcium. In congenital syphilis the results vary, depending on the presence or absence of infection of the nervous system; in about 33 per cent of the author's cases the central nervous system was involved. In the author's cases of chorea, the cerebrospinal fluid was normal.

Meningism, namely, meningeal symptoms without meningitis as indicated by the spinal fluid, is found in children more often than in adults, since the meninges of children react more frequently to infectious diseases, such as pneumonia, mumps and scarlet fever. In postdiphtheritic paralysis, the fluid is clear, the cell count is within normal limits, and there is a tendency for an increase in albumin proportionate to the amount of involvement of the nervous system.

In acute anterior poliomyelitis the fluid varies with the stage; in the paralytic stage, the pressure is increased, the cells vary between 50 and 150 per cubic millimeter, mostly lymphocytes, the amount of globulin is increased and the Lange curve is about the same as in the preparalytic stage; in the stage of recession the fluid gradually returns to normal, a process requiring from two weeks to two years.

In epidemic encephalitis the pressure is increased, the globulin is increased, the amount of sugar is occasionally increased (thus serving in the differentiation from meningitis in which the amount of sugar is usually decreased) and the Lange test often shows a discoloration in the syphilitic zone. A type of encephalitis may occur in which there is no meningeal reaction and in which the cerebrospinal fluid is normal in every respect. In meningitis during infancy and childhood, the fluid presents the same changes which occur in meningitis during adult life.

VONDERAHE, Cincinnati.

EXOGENOUS FACTORS IN SCHIZOPHRENIC PSYCHOSES. M. FISCHER, Arch. f. Psychiat. **83**:779 (May) 1928.

Fischer discusses the occurrence of typical schizophrenic reactions apparently due to organic diseases of the central nervous system, and the light that such occurrences may throw on the study of the etiology of schizophrenia in general. Two cases are reported in which injuries to the skull and the brain were followed by schizophrenic psychoses. In case 1, a man, aged 32, who did not show any abnormal behavior other than heavy drinking, sustained an injury in the left temporal region; an operation was performed and a hematoma removed from over the temporal lobe extending to the base of the brain. He made a good recovery, although the epileptiform convulsions which were present before the operation recurred on several occasions following it; they then disappeared entirely. A short time after the operation, he began to hear whispering in the right ear; following this, he developed grandiose ideas with definite hallucinations. About two years after the operation, he returned to the hospital; following this, there was a progressive deterioration with the persistence of the delusions and hallucinations. In case 2, the patient, aged 31, who had been normal until the accident, fell from a second story window and struck his head. He was unconscious for two days, and a diagnosis of concussion of the brain was made. Following apparent recovery there was at first an increased irritability and "nervousness," and, about two years after the accident, a sudden development of a schizophrenia-like psychosis with hallucinations, delusions and progressive deterioration. In case 3, a man, who had been normal up to the age of 36, was admitted to the hospital with neurosyphilis and diabetes. While in the hospital, he developed delusions and hallucinations, mostly of a depressive nature. Following a course of treatment with malaria, he recovered from the diabetic condition, but retained a typical schizophrenic mental reaction.

In addition to these three cases, the author reports three so-called post-traumatic psychoses, in which, however, the trauma can be regarded as only a precipitating cause. In the last two, there were indications of psychotic episodes before the trauma. The author discusses his observations and concludes that typical schizophrenic reactions may occur on the basis of organic lesions in persons who had not been mentally abnormal previously; one should be careful, however, not to overestimate the frequency of these reactions and not to ascribe to organic diseases the psychoses that occur in persons who were mentally abnormal before the injury. The fact that such reactions do occur on an organic basis, however, should encourage one to search for an organic etiologic factor in cases that superficially do not show signs of it.

MALAMUD, Foxborough, Mass.

DEGENERATIVE MYELITIS FOLLOWING SPINAL ANESTHESIA. NONNE and DEMME, Wien. klin. Wchnschr. **41**:1002, 1928.

Since the introduction of spinal anesthesia, numerous reports have appeared in the literature of serious sequels. Occasionally, sacral decubitus and necrosis occur. Even more common are transient eye palsies and respiratory disturbances. The following case is reported as the patient died six months after the spinal anesthesia, and a complete pathologic study was possible.

The patient, a man, aged 51, was operated on under spinal anesthesia (tutocain) for bilateral inguinal hernia, in September, 1926. Following the spinal injection, the patient noted difficulty in the movement of his legs; within three days, decubitus occurred on the buttocks. On the same day that the operation was performed, two other patients received spinal anesthesia from the same preparation; in both there was transitory sensory, motor and vesical impairment.

Examination of the patient at the time of Nonne's observation showed a marked transverse myelitis extending from the fourth lumbar segment downward, and involving both motor and sensory functions. The patient died six months after the operation.

At autopsy, there were a severe pyelocystonephritis, pulmonary tuberculosis and tuberculosis of the colon. Microscopically, the lumbar and sacral cords showed evidence of an inflammatory change. The meninges were thickened. The most severe changes were present in the conus and cauda. The posterior portion of the conus showed an extensive degeneration, and the posterior columns were severely affected. All the caudal roots showed a marked degenerative change. Ascending the cord, the degeneration was confined to the column of Goll.

A case of myelitis following the intraspinal injection of arsphenamine, reported by Nonne, showed a degenerative myelitis, without evidence of any inflammatory change. Cases, however, have been reported in which inflammatory changes have occurred following injections of arsphenamine. In the present instance it appears that the degenerative process was of a toxic, degenerative character, without any special inflammatory change. The interesting feature in the present report is the fact that, on the same day, two other patients sustained transitory changes in the cord. A careful investigation of the preparation of the anesthetic did not reveal faulty technic, and impurities were not discovered.

From the standpoint of the authors, there appeared to be no question that the myelitis was directly the result of intraspinal medication, and they properly asked the question whether it is wise, in minor surgical procedures, to subject the patient to an intraspinal injection.

MOERSCH, Rochester, Minn.

THE MYOSCLEROSIS OF OLD AGE AND ITS RELATION TO AKINETO-HYPERTONIC SYNDROMES. JEAN LHERMITTE, Encéphale **23**:89 (Feb.) 1928.

It has been shown definitely that a motor weakness of the inferior members can exist which depends exclusively on muscular alterations and is not due in any way to lesions of the cerebrospinal or peripheral nerve. At the outset this is a diffuse amyotrophy with flaccid paraplegia, and it may progress to the end as the same type. This condition, described by Vulpian and Charcot, is asserted

to be marked purely by a degeneration of striated muscle fibers and their transformation into adipose tissue. Furthermore, such a fatty degenerative change may extend to the viscera.

This malady affects the female sex much more frequently than the male. Its clinical onset is marked by pain, cramps and localized twinges. The characteristic attitude of lateral decubitus develops fairly rapidly: the thighs are flexed on the abdomen, and the legs flexed on the thighs; often the legs are crossed, and the entire body is almost immobile because of the pain provoked by motion. Reflexes are often not obtainable because of the attitude; when elicited, they are found to be diminished; the plantar reflex is invariably normal. There is not any great alteration in electrical response, but mechanical irritability is often augmented and myo-edema is constant.

Other conditions are described, however, which may present diagnostic difficulties in separation from the myosclerosis already described: (1) Foerster's syndrome or arteriosclerotic rigidity in which the attitude is similar but in which certain parkinsonian symptoms should be in evidence; (2) senile muscular rigidity, which A. Jacob describes, and which like the arteriosclerotic type of rigidity, involves a mental state, often amounting to dementia. On comparison, Lhermitte consigns both conditions to the same category. He then presents a case which shows association of the retractile myosclerosis with arteriosclerotic rigidity. Finally, he shows that retractile myosclerosis may complicate a paraplegia of cerebral origin.

On the other hand, since myosclerosis does appear in what would seem to be an uncomplicated state, the seeming overlap of such diverse conditions must be explained. It may be that this pure symptomatology is only apparent, Lhermitte observes, and that the absence of pallidal rigidity, for instance, can be explained as a "masking" due to the marked structural alterations in the voluntary muscles. If this hypothesis is correct, myosclerosis could be looked on less as an autonomous condition than as a complication accompanying several types of hypertonia.

ANDERSON, Kansas City, Mo.

VASOMOTOR EFFECTS OF STIMULATING THE RIGHT SPLANCHNIC NERVE. J. H. THOMPSON, J. Physiol. **65**:441 (Aug.) 1928.

Thompson studied the effect of stimulation of the right splanchnic nerve on the form of the blood pressure curve. With the intact animal under anesthesia, or the decerebrate animal, the splanchnic nerves were exposed in the abdominal cavity and divided, and the peripheral ends were stimulated, using an induction current rapidly interrupted by a Neef hammer. It is important to note that the stimulations were made at short intervals, that is, as soon as the blood pressure returned to the mean, the next stimulus was given. The stimulus had to be adequate.

The first stimulation usually effected a rise of blood pressure, identical with that obtained from the left nerve, except that there was a distinct tendency for the blood pressure to rise at the end of stimulation no matter at what position in the curve it ceased. With later stimulations, however, a marked difference became apparent: (1) After about four successive and equal stimulations, at short intervals, it was impossible to obtain any rise of blood pressure on the application of the same strength of stimulus. Apparently, there was a rapid exhaustion of the vasoconstrictor fibers in the nerve, the exhaustion being uniform. Even after thirty stimulations of the left nerve, such exhaustion cannot be produced. (2) Coincident with the diminution of the rise after the first stimulation, a remarkable kind of rise was found to occur at the moment when stimulation was concluded. It consisted of a rapid rise with a gradual decline. Tying the suprarenal glands or clamping the superior mesenteric or portal veins did not affect it. (3) Further stimulation of the right nerve with the same strength and frequency of current, after exhaustion of the rises had occurred, resulted in a pure fall of blood pressure.

Ultimately, the strongest stimulus elicited falls, and it became impossible to obtain pressor responses. A curious phenomenon constantly observed by Thompson

was that the depth of the fall was inversely proportional to the strength of the stimulus. Sometimes if an interval of rest was given to the nerve, the effect of exhaustion leading to a fall on stimulation disappeared temporarily, and was substituted for by a rise; but if the nerve was stimulated frequently and many times, no such recovery was experienced although two hours were allowed to elapse.

Thompson concludes that the right splanchnic nerve contains not only vasoconstrictor fibers, but also a considerable number of vasodilator fibers. Allusion is made to certain anatomic differences between the right and left nerves.

ALPERS, Philadelphia.

ENCEPHALOMYELITIS IN VIRUS DISEASES AND EXANTHEMAS. HUBERT M. TURNBULL, Brit. M. J. **2**:331 (Aug. 25) 1928.

The pathologic observations in cases of postvaccinal encephalomyelitis are usually uniform but depend to some extent on the duration of the illness. In the first stage, congestion and edema are present, with hemorrhage into the leptomeninges, punctiform hemorrhages into the spinal cord, and sometimes blurring of the normal line of demarcation between gray and white matter. The lesions extend from the cortex to the sacral cord and vary in their severity; occasionally, they are greatest in the midbrain. In the cerebrum the white matter is more severely affected, whereas in the brain stem and spinal cord the gray matter suffers more. During the acute stage, slight infiltration of the pia with mononuclear elements, and congested and thrombosed vessels with occasional hemorrhages are found. The most characteristic observation in the parenchyma is the presence of perivascular zones of demyelinization which are present, apparently, in all cases. They are sharply defined and are associated with disappearance of the axis cylinders and slight hyperplasia of the neuroglia. There is a moderate infiltration of the demyelinated areas by microglia phagocytes which contain fat and fragments of myelin. Polymorphonuclear leukocytes are rarely encountered. There is an inconspicuous infiltration of the vascular adventitia. The nerve cells are well preserved, although Bastiaanse has described neuronophagia in the locus niger. In the subacute type the perivascular sheaths are "distended with a mosaic of large fat-granule cells." These cells are also found in the meninges.

The author points out that such observations sharply distinguish postvaccinal encephalitis from poliomyelitis and encephalitis lethargica, and that it belongs to a group of conditions characterized by demyelinization. The chief examples of the condition are disseminated myelitis of Westphal, disseminated sclerosis, and Schilder's disease. Conditions resembling postvaccinal encephalitis have been observed independently of recent vaccination and even of observed exanthems, but the disease has been known to follow scarlet fever, measles, smallpox and other diseases. Even in the paralysis complicating antirabic treatment the histologic condition seems closely related to this type of encephalomyelitis. If there are certain points of differentiation, the resemblances of the histologic picture in each of these conditions warrant the hypothesis that the pathogenic agent is essentially similar and the histologic differentiation probably artficial.

FREEMAN, Washington, D. C.

PARALYSIS OF ALL FOUR LIMBS CURED BY REMOVAL OF A SPINAL TUMOR. WALTER BROADBENT and G. W. BERESFORD, Brit. M. J. **1**:1063 (June 23) 1928.

Fourteen months previous to admission to the hospital, the patient, a woman, aged 51, noticed numbness in the right hand. Six months later she tripped and fell, after which she began to lose power in the right arm. This progressed and in three months she was dragging the right foot, could not work and had aching pains in the right shoulder and arm. Two months later, the right hand had become useless, the right leg was so involved that she could not walk and the left leg was affected. A month later, the right limbs were paralyzed; she could move the left leg only slightly, and there was weakness in the left shoulder and

hand. Both trapezii were wasted but not the arm muscles. The reflexes were increased. She complained of intense headache, pains in both shoulders and "pins and needles" all over the body. There was anesthesia of the right hand and forearm and diminished sensation in the same area on the left arm. Sensation over the legs and body was less than normal, and distinction between heat and cold was not accurate. There was constipation but no incontinence.

The neck was freely movable and x-ray examination did not show any disease of the bone. The cerebrospinal fluid obtained from lumbar puncture was not under pressure. It contained considerable excess globulin and the colloidal gold curve was 5554433221; there was no increase of cells with a normal reduction of Fehling's solution. The Wassermann reaction was negative. The fluid from a cistern puncture contained only a faint trace of globulin; it did not contain cells. The colloidal gold curve was 0111000000. Iodized oil 40 per cent, when injected, stopped opposite the level of the first and second cervical vertebrae. In an operation, the dura in the region of the second, third and fourth cervical vertebrae felt hard and was not pulsating. On opening it, a tumor was found in the postero-lateral aspect, pressing into the right side of the cord. This was attached by a pedicle to the dura. Its appearance was rather like a large raspberry, fleshy to feel and not very vascular. Microscopic examination showed it to be an endothelioma.

After the operation, movements of the left arm soon began to improve, then those of the left leg. It was nearly a month before the right leg showed signs of recovery and even longer for the right arm. After ten weeks, the patient could walk with a little assistance, spasticity not having quite disappeared. Movements of the left arm and hand were perfect, and the patient could lift a glass to her mouth with the right hand.

PETERSEN, Montreal.

INOCULATION OF MALARIA IN NEUROSYPHILIS. PHILIP B. MATZ, J. Nerv. & Ment. Dis. **68**:113 (Aug.) 1928.

The true nature of the mechanism involved in malarial therapy is not known. A nonspecific stimulation by the malarial infection, accompanied by hyperemia and increased permeability of the capillaries of the brain, is generally believed to be the cause of the reaction. In the United States Veterans' Bureau, the method of treatment in a series of 346 patients was the intravenous injection of about 2 cc. of malarial blood. The cases of patients who had well defined cardiovascular diseases such as nephritis and tuberculosis were excluded. Each patient was allowed to have approximately twelve paroxysms and was then placed on quinine. This was followed by antisyphilitic treatment.

The period of incubation varied from one to nineteen days; the average period of incubation was six and one-half days. The highest temperature reported was 42 C., the lowest 36.7 C.; the average temperature was 39.4 C. Seven paroxysms was the smallest average number reported in the patients inoculated; 5.4 per cent of the patients failed to develop the infection and were considered immune to the malarial plasmodium. The longest period during which patients at the Bureau were observed was forty-seven months; the average period of observation for the whole series was twenty-seven months.

The complications noted by the observers at the Bureau were: mild jaundice, nausea, vomiting, loss of appetite, loss of weight, edema of the ankles, anemia, incontinence of urine and feces, convulsive seizures, delusions and restlessness. Most of the patients lost weight during the treatment, but regained a little after the cessation of the paroxysms. The average gain in weight subsequent to treatment was 17.4 pounds (7.8 Kg.). The manic and expansive types of paresis were more amenable to treatment than the demented type. The condition in the patients at the Bureau was diagnosed not only as paresis but as taboparesis, cerebrospinal syphilis and tabes. Twenty-three and four tenths per cent of 346 patients treated were greatly improved; 41.04 per cent were improved; 22.83 per cent remained unimproved; 7.5 per cent became deteriorated, and 3.47 per cent died.

In 81.2 per cent of a series of 279 cases under observation the cytology of the spinal fluid was benefited, following this treatment. In 72.04 per cent of the cases,

the Wassermann reaction of the blood became negative or was modified; in 69.89 per cent, the test for globulin gave negative results or showed a reduced amount; in 59.85 per cent, the Wassermann reaction of the spinal fluid became negative or was modified, and in 54.48 per cent, the colloidal gold curve was modified.

HART, Greenwich, Conn.

COMMUNICATING HYDROCEPHALUS (SO-CALLED IDIOPATHIC HYDROCEPHALUS). J. H. GLOBUS, Am. J. Dis. Child. **36**:680 (Oct.) 1928.

The author distinguishes between obstructive hydrocephalus, caused by a block of the normal outflow of fluid from the ventricular cavities and produced by tumor, ependymitis, atresia of the aqueduct, etc., and communicating hydrocephalus, in which all the ventricles are in free communication with the subarachnoid space around the spinal cord. The studies of Dandy are quoted at length; the cause of communicating hydrocephalus is found primarily in the closure of many or all of the subarachnoid channels over the brain surface, a condition produced by meningitis, developmental anomalies, and neoplasms so situated as to obstruct one or more cisterns at the base of the brain. Anything which will interrupt the free flow of the cerebrospinal fluid to the large subarachnoid space will bring about an excessive accumulation of fluid in the ventricles with consequent distention.

Five cases which came to autopsy were studied by the injection of India ink before the brain was removed, and by histologic examination. Case 1 showed a developmental anomaly in which the subarachnoid channels failed to form; the arachnoid near the cisterna magna was still united with the pia, the two forming a solid mass of mesenchymal tissue as found in the primitive meninx. Case 2 was similar. The retardation in the histogenesis of the pia-arachnoid in this case was associated with another developmental anomaly in the form of a spina bifida. Case 3 resembled cases 1 and 2, but the hydrocephalus developed more slowly and was characterized by a greater extent of absorbing surface in the subarachnoid as shown by the test with India ink and by histologic examination. In case 4 an internal hydrocephalus was associated with meningococcus meningitis; when India ink was injected into the cisterna magna prior to opening the skull, it was found subsequently in the cisterna magna, cisterna interpeduncularis, and a limited area of the cerebellum, but none reached the surface of the cerebral hemispheres. It did flow into the ventricles, however, indicating the patency of the foramens. In case 5, there appears to have been a subarachnoid hemorrhage following a fall, with resultant productive changes in the meninges leading to obliteration of many of the subarachnoid channels. The author does not think that the two types can be distinguished with certainty by clinical methods.

VONDERAHE, Cincinnati.

NERVE EXCITATION BY MULTIPOLAR ELECTRODES. W. A. H. RUSHTON, J. Physiol. **66**:217 (Nov.) 1928.

In this paper the author presents a further range of observations in support of his original assumptions to explain the changes of the threshold for nervous excitation which are observed when certain alterations are made in the position of the electrodes. The assumptions were, essentially, that the nerve might be considered as a cylinder with a resistant sheath and a conducting core, and that the current which excited was the portion which left the core through the sheath. The experiment is performed with an arrangement of tripolar electrodes with a single straight stretch of nerve, and lends itself to greater accuracy than those with a bent nerve such as was used in the experiments previously reported.

Rushton found that the prediction of the formula obtained in his former work is fulfilled in the cases tried in this new series; namely, tripolar electrodes which are polarizable, lightly chlorinated and heavily chlorinated; and quadripolar electrodes which are nonpolarizable.

The author believes that in one respect the experiments give a definite result. If the cylindrical membranes (the membranes of Nernst) are to have any place in the visible histologic structure of the nerve, it seems likely that they must be located as bounding the medullary sheath either inside or outside, and hence the stimulating current will have to pass through this resistant sheath to excite. The amount of current which will pass through a resistant sheath of this kind will depend on the applied potential all along the nerve and may be worked out from the physical principles underlying current distribution. This has been done, and the results have been applied to several kinds of potential distribution. In every case it has been found that, in order to obtain a threshold excitation, the values of the potential at various points along the nerve have to be so adjusted experimentally that the current leaving the sheath at the point where excitation is occurring remains always at the same value. This, on the one hand, supports the suggestion that the excitable membranes are in close proximity to the medullary sheath so that the current passing through the membranes is the same as that passing through the sheath, and it affords strong evidence that one is right in supposing that the membranes are cylinders coaxial with the nerve. On the other hand, it supplies a simple and comprehensive explanation of the variation of threshold with the position of the electrodes.

ALPERS, Philadelphia.

THE SIGNIFICANCE OF AFFERENT IMPULSES FROM THE SKIN IN THE MECHANISM OF VISCERAL PAIN; SKIN INFILTRATION AS A USEFUL THERAPEUTIC MEASURE. SOMA WEISS and DAVID DAVIES, Am. J. M. Sc. **176**:517 (Oct. 28) 1928.

Various views on the mechanism of visceral pain are discussed, particularly those of Head and Mackenzie; these are essentially the same; painful impulses from a viscus pass along the splanchnic nerves to certain segments of the cord and cause the development of an irritable focus. Normal impulses arising from the skin and muscles enter this abnormally irritable segment, and, because of the increased irritability, give rise to painful sensations which are referred to these peripheral structures. Another theory is that a special stimulus is needed; this precludes direct stimulation of a viscus in producing pain experimentally because of the possible inadequacy of the stimulus.

The problem of experimental proof is approached from a new angle; if one accepts the views of Head and Mackenzie, cutting off the normal impulses of the skin from the area of referred pain would stop the pain. Weiss and Davies present twenty-five cases of various types, with referred pain, in which infiltrations of procaine hydrochloride were made into the skin in the involved area. In only one case was there no relief; in three cases, there was partial relief, and in the remainder, complete relief for periods of from twenty-five minutes to six hours. Similar results were recorded in two cases of induced referred pain. The authors believe that the relief was not induced by a systemic effect of the drug, as similar amounts introduced in other areas did not have any effect. In certain cases of pain associated with vomiting, infiltration produced relief from the pain but did not have any effect on the vomiting; consequently, the sympathetic reflex arc must have remained intact. It is believed that these results support the theory of referred localized pain; also that localized referred pain resulting from disease of a viscus may be partially or completely relieved temporarily by infiltration of the involved area with a local anesthetic.

WAGGONER, Philadelphia.

TUMOR OF THE BRAIN SIMULATING ENCEPHALITIS LETHARGICA. S. MCCLEMENTS, Brit. M. J. **1**:1061 (June 23) 1928.

A man, aged 57, had a mild attack of influenza in April, 1926, from which he completely recovered during the following month. Four weeks later, he began to grow drowsy and lethargic; in a few days, his vision for near objects became dim. He grew worse for four weeks, developing diplopia; his temperature varied from 98 to 100 F. In July, he improved, and became brighter. The diplopia

disappeared. Except for spasmodic twitching of the legs he had almost regained his health in September. In October and December, 1926, and April, 1927, he had relapses followed by marked improvement. Each attack, however, left him a little more stuporous. In June, 1927, he became markedly lethargic. When awakened, his mental condition appeared clear and he was able to answer questions in a rational manner. His face was smoothed out and expressionless; there was ptosis, a monotonous tone of voice and weakness of the right sixth nerve. His vision for near objects was dim; the right pupil did not react to light and the left reacted sluggishly; accommodation was normal. There was a well marked papilledema of the right disk, and the retinal veins on the left were swollen. He died at the end of November, 1927. Autopsy revealed a spherical tumor, 2 inches (5 cm.) in diameter and gelatinous, lying in a smooth-walled cavity in the right frontal lobe. Posteriorly, between the tumor and the wall of the cavity, there was a clot of blood 1 inch long and a quarter of an inch broad (2.5 by 0.63 cm.).

A Case of Progressive Muscular Atrophy of the Peroneal Type. George Parker, Brit. M. J. **1**:1062 (June 23) 1928.

The familial aspect of this disease has been pointed out by several writers. The condition is transmitted both in the male and female line. There are instances in which none of the relatives are affected and others in which several siblings become affected simultaneously, for the first time in the family. Sometimes it appears after a febrile illness such as measles. It may commence in childhood, but more often in the second or third decade. Generally, the first wasting is seen in the feet or peronei on each side, but occasionally it appears first in the hands and forearms. The weakness gradually spreads to the upper segments of the limbs. The trunk and face are rarely affected. Bulbar symptoms are unknown. Reaction of degeneration can be found in some of the muscles in practically every case, while there may or may not be cramps, pains, coldness and lividity of the skin, fibrillary tremors or slight sensory changes. The author reports a case with wasting of the distal segments of the limbs and loss of power, beginning at the age of 19 years and steadily progressing. The reflexes are diminished, and reaction of degeneration is present. Eleven males and ten females of the family are similarly affected. The first recorded case was that of the patient's great-grandfather of whose fifty-four descendants twenty-one are already affected; the others are not yet old enough to develop the disease. The condition seems to be transmitted more readily through the male than through the female line, and it has little effect on the duration of life.

Duplication of the Spinal Cord. F. Parkes Weber, Brit. M. J. **1**:1106 (June 30) 1928.

The patient, a man, aged 51 years, was admitted to the hospital with a spastic paretic-ataxic gait and signs suggesting combined degeneration of the spinal cord. The liver and spleen were enlarged; there was no free hydrochloric acid in the stomach contents, and the Wassermann reaction was negative. The red blood cells numbered 3,392,000; the white cells 17,000; the hemoglobin was 70 per cent. There was a little irregular fever and apparently a remnant of pneumonia in the central portion of the right lung, which probably explained the leukocytosis. The patient had first noticed something wrong with his feet in August, 1927; weakness of the legs developed about March 19, 1928, and he was admitted to the hospital ten days later. In the hospital, the weakness increased in spite of liver diet, etc.; the patient developed paradoxic incontinence of urine and cystitis, with bedsores, and death occurred on April 26.

Necropsy and microscopic examination confirmed the diagnosis. During the macroscopic examination, however, it was noted that, close along the left side of the lower part of the spinal cord, there was another cordlike body of the same consistence as the cord but smaller (about 1 cm. in diameter) and without nerve roots. Its upper end arose from the substance of the cord by a base somewhat over 1 cm. in diameter, the uppermost part of the insertion being 7.5 cm. above

the terminal point of the conus. At its lower free end, it tapered to a point which was 1.5 cm. above the lower end of the conus. Microscopic examination showed this to be a fairly well-developed duplicate cord, with a central canal, etc. This duplicate cord showed the same distinct signs of combined degeneration as the main spinal cord.

THE ETIOLOGY OF MONGOLISM. HUBERT ARMSTRONG. Brit. M. J. **1**:1106 (June 30) 1928.

Is mongolism inherent in the unfertilized ovum or is it acquired after fertilization? The idea that mongolism is imparted to the ovum in fertilization is not so tenable since evidence appears to point to the maternal factor so constantly. An argument in favor of the postfertilization embryonic theory is that mongols are so often the subjects of other congenital abnormalities. It is argued that the mongolian characteristics originate during the first six weeks of intra-uterine life. On the other hand, an ovum which already contains an inherent defect is more likely to develop other abnormalities than one which does not. David Greig said that mongolism "seems rather to be a defect in growth (foetal) than a defect in development (embryonic)." He stated that "even from the skull alone there seems evidence that development normally begun has mapped out all the structures and features in the embryo, but has failed to lead them to perfection during foetal growth . . . this failure in the bones is accompanied or followed by defective growth elsewhere, notably in the nervous system."

Armstrong believes that whatever the almost undeniable maternal influence may be, it is exercised on the ovum during its maturation, and therefore before fertilization has followed and segmentation started. The chief argument in favor of this theory is the occasional occurrence of twins, one of whom is a mongol and the other not. There have been about three known instances of twins who were both mongols. The original records of these cases, however, are so inaccessible that although the twins were of the same sex, complete proof that they resulted from uniovular pregnancies is wanting. The fact, however can be assumed. There are also three cases on record in which the twins were of the same sex, one being mongol, proof of uniovular pregnancy is lacking here also. The records, however, do not show an instance of mongolism in twins of different sex.

PETERSEN, Montreal.

TWO CASES OF THE SYNDROME OF VAN DER HOEVE: BLUE EYES, BRITTLE BONES AND DEAFNESS. F. TERRIEN, P. SAINTON and P. VEIL, Ann. d'ocul. **165**:58 (Jan.) 1928.

The mother was exceedingly deaf, had blue eyes and had had several spontaneous fractures. She had five brothers and sisters: Two of these had blue eyes; one was deaf and had never had fractures, and another heard well but had already had three fractures. A daughter, aged 12 years, presented the same signs as the mother, and in addition showed abnormal laxness of the articular ligaments. The oculocardiac reflex was inverted in the mother and daughter. The conditions suggest a possible endocrine-sympathetic origin or a parathyroid dysfunction, since the parathyroid plays an important rôle in the metabolism of calcium.

A CASE OF VOLUNTARY NYSTAGMUS OF BOTH EYES. MERIGOT DE TREIGNY, Ann. d'ocul. **165**:55 (Jan.) 1928.

Merigot de Treigny saw a patient with voluntary horizontal nystagmus of the ocular type.

VOLUNTARY NYSTAGMUS . COLOMB, Ann. d'ocul. **165**:427 (June) 1928.

A case of voluntary nystagmus in a man, aged 25, is reported by Colomb and compared with fifteen other cases reported in the literature. Without moving the eyelid or any of the facial muscles, the patient was able voluntarily

to produce rapid, equal, horizontal oscillations with an amplitude of from 1 to 2 mm., at the rate of approximately 200 per minute. The family history showed that one brother also had voluntary nystagmus. The conclusion is reached that voluntary nystagmus is produced centrally by an act of will which sometimes leaves a sensation of fatigue or a vague pain, accompanied at times by other spasms. Enlargement of the palpebral fissure has been noted; myosis with deformation of the pupil has been reported by Weekers, and spasm of accommodation, by Mauersberg.

BERENS, New York.

ANALOGIES BETWEEN THE AQUEOUS HUMOR AND THE CEREBROSPINAL FLUID. Editorial, J. A. M. A. **89**:2265 (Dec. 31) 1927.

One result of the present tendency to attempt to test physicochemical explanations of bodily physiologic changes has been to remove certain characteristic physiologic processes from the category of the more vague manifestations designated as secretion and to class them rather with other clearly recognized instances of simple fluid exchanges. When two fluids are separated in the body by a membrane, their exchange will follow the well known principles of diffusion and equilibrium long recognized in biology. However, many persons have considered these principles inadequate to explain certain instances of fluid exchange. The aqueous humor of the eye was attributed to secretion by the ciliary process, and the cerebrospinal fluid was alleged to be secreted by the choroid plexus. Recent evidence has tended to show that the cerebrospinal fluid is in actual equilibrium with the blood, and that its changes in pressure as well as its composition can be explained by simple fluid exchange.

CHAMBERS, Syracuse, N. Y.

THE IDEA OF UGLINESS. M. NATHAN, Encéphale **23**:748 (Sept.-Oct.) 1928.

Two cases are quoted from the works of Janet, in which young women became obsessed with ideas of personal ugliness, these ideas coloring their entire lives up to the point of treatment and cure. Then follows a detailed account of a case observed by the author—a girl, aged 20, who had developed similar ideas. These dated back to the age of 5, when basic inferiority feelings were inculcated as a result of jealousy of a younger sister. The parents, failing to understand the situation, built up in the patient definite ideas of moral reprobation, using such terms as "bad girl" and "ugly girl" to voice their displeasure. From this grew beliefs in a lack of physical beauty, which eventually, at beginning adolescence, became a real obsession. In all these cases, ideas of guilt, culpability and inferiority appear to be the fundamental difficulties, the particular seizing on lack of beauty being more or less incidental. Naturally, there need be no real lack of physical comeliness—it rests entirely in the idea.

ANDERSON, Kansas City.

THE ELECTRIC RESPONSE OF A NERVE TO TWO STIMULI. YNGVE ZOTTERMAN, J. Physiol. **66**:183 (Oct.) 1928.

Zotterman endeavored to confirm the results obtained when a nerve is stimulated at various frequencies by observations on the total electric response to two stimuli only with various intervals between them. He arranged his preparation so that the action potential of a single nerve impulse could be read directly on the scale of a sensitive, high resistance, Downing galvanometer. Fifteen reliable experiments were performed on fifteen different nerves, with intervals between the shocks increasing from one and four-tenths to thirty seconds and returning. The action potential returns gradually to its full value as the interval after the preceding shock is increased. This return of the action current to its full value is relatively slow, being incomplete in fifty seconds. After a stimulus the return of the energy in the action potential of a second impulse follows a similar curve to the return of the production of heat.

ALPERS, Philadelphia.

SWIFT-FEER ACRODYNIA: ITS RELATIONSHIP TO EPIDEMIC NEURAXITIS. L. VAN BOGART, J. de neurol. et de psychiat. **28**:118 (Feb.) 1928.

A case of acrodynia is described in an adult who developed a neurodermatologic syndrome characterized by an extreme asthenia, considerable pain of the skin and muscles, an intense prurigo, trophic ulcers and generalized muscular hypotonia. To this visceral symptoms were added: spasmodic constipation, gastric symptoms, arrhythmic tachycardia and hypotension. A lumbar puncture disclosed hyperalbuminosis and hypoglycorrhachia. The author is inclined to consider acrodynia as a syndrome of infectious origin involving electively the sympathetic system. He is unwilling to accept the identity with epidemic encephalitis which such investigators as Pierre Janet and T. Knifer have established. The absence of myoclonia and of ocular disturbances and the dissociation between hyperglycemia and hypoglycorrhachia in the majority of cases contribute to this differentiation.

FERRARO, New York.

THE FORM AND MOVEMENTS OF THE PUPIL IN VERTEBRATES. P. BRETAGNE, Ann. d'ocul. **165**:204 (March) 1928.

Bretagne does not know of any vertebrate in which the eye is without iris or pupil. Most of the irides, but not absolutely all, contract under the influence of light. They may be round or oval according to the species, and in dilatation and in contraction they may remain round, but in some animals the form varies, and they appear as vertical slits, horizontal openings or in the form of a cross. He considers the cause of these different forms of contraction with relation to the needs of the particular animal. He points out that the retina is rudimentary in some forms, is sensitive to light, but only slightly capable of visual acuity. The contraction of the pupil depends more on the amount of light than on the perfection of the retinal image, and the whole problem revolves about the conditions under which vertebrates live.

BERENS, New York.

ENCEPHALOMYELITIS IN VIRUS INFECTIONS WITH EXANTHEMAS. JAMES MCINTOSH, Brit. M. J. **2**:334 (Aug. 25) 1928.

McIntosh reports the reproduction in rabbits, by means of ordinary vaccinal virus, of a fatal encephalitis which showed the characteristic histologic features of postvaccinal encephalitis in man. He thus carried Levaditi's work a step further, since the French author failed to obtain uniform results in rabbits. McIntosh reports that, by means of intravenous injections of Levaditi neurovaccine and of his own highly virulent vaccine, he produced focal lesions in the internal organs of rabbits closely resembling those found in patients with postvaccinal encephalitis and similar to the focal lesion that occurs in true smallpox. This excludes the hypothetic virus which was thought to contaminate the vaccinal virus and produce encephalitis in these cases.

FREEMAN, Washington, D. C.

DIAGNOSIS OF SYPHILIS FROM THE STANDPOINT OF THE INTERNIST WITH SPECIAL REFERENCE TO THE INCIDENCE OF NEUROSYPHILIS OVER A PERIOD OF TEN YEARS. WILLARD C. STONER, Am. J. Syph. **12**:340 (July) 1928.

The author's personal observations indicate that approximately 5 per cent of the white population of his community (Cleveland) are syphilitic; these observations extend over a period of ten years and show a much lower incidence during the last five years. Neurosyphilis is probably less common than formerly, as shown by a much smaller incidence in the last five-year period when a larger volume of cases for diagnosis were studied than in the first five-year period. A survey of admissions to hospitals over the country for a ten-year period indicates a definite lowering of the incidence of neurosyphilis.

ANDERSON, Kansas City.

A Case of Intracranial Hypertension Without Papilledema, Detected by Hypertension in the Retinal Arteries. M. Kalt, Ann. d'ocul. **165**:692 (Sept.) 1928.

Kalt examined a woman, aged 40, who complained of headache and vertigo. General neurologic and otologic examinations gave negative results. Examination of the eye gave negative results except that pressure in the retinal arteries was markedly elevated; in the right eye it measured 130/60, and in the left, 130/70. Pressure in the brachial artery was 155/95. Normally, the retinal diastolic pressure should be a little less than half of the brachial diastolic pressure when the general blood pressure is normal. When the general blood pressure is raised, the retinal diastolic pressure is usually half of the brachial diastolic pressure. A lumbar puncture revealed a marked increase in the pressure of the cerebrospinal fluid.

Berens, New York.

Some Therapeutic Attempts with African Recurring Fever in Mental Diseases. F. D'Hollander and E. de Greff, J. de neurol. et de psychiat. **27**:555 (Oct.) 1927.

The authors have used *Spirochaeta Duttoni*. They have injected the blood of mice previously infected, or a pure culture of the spirochete. They have also injected human beings subcutaneously with the blood of a previously infected patient. The period of incubation was from seven to eight days. The average number of attacks was three. Immunity to a second inoculation developed. Sulpharsphenamine injected intravenously was used to stop the course of the fever if that was deemed necessary. Five patients with melancholia and fifteen with dementia praecox were treated. The results were practically a failure as appreciable changes did not occur. The course of the disease seems to have been the same as in patients that had not received treatment.

Ferraro, New York.

Arterial Encephalography and the Diagnosis of a Tumor. E. Moniz and A. Lima, Encéphale **23**:195 (March) 1928.

The injection of 25 per cent sodium iodide into the internal carotid is accomplished without inconvenience to the patient, according to these authors. A case is described in which such an injection was made after death and demonstrated important modifications whereby diagnosis was made of the presence of a tumor, which was verified at a subsequent autopsy. Three aspects of encephalography by this method aid in deciding tumor localization: the displacement of the cerebral arteries; the stasis of cerebral fluid in highly vascularized tumors, and the existence of new formations of arterial networks.

Anderson, Kansas City.

Clinical Results from the Use of Bismarsen in Tabes Dorsalis. Norman Thomas, Am. J. Syph. **4**:536 (Oct.) 1928.

Twenty patients showing the clinical and serologic signs of tabes dorsalis were treated with intramuscular injections of bismarsen. A decrease in lightning pains and an average gain in weight of 9 pounds were observed, ataxia being influenced to a lesser extent. The effect on the Wassermann reaction of the blood was more marked than on that of the spinal fluid. A reversal to negative serology occurred in three cases. The results appear more promising in early stages, but the author considers it too soon to evaluate bismarsen in antisyphilitic therapy.

Jenkins, Philadelphia

BILATERAL HEMIANOPIA WITH CONSERVATION OF MACULAR VISION, AS A RESULT OF A TUMOR OF THE LEFT OCCIPITAL LOBE. D. MANOLESCO, Ann. d'ocul. **165**:215 (March) 1928.

Manolesco reports a case of sarcoma of the left occipital lobe in a patient, aged 19 years, who was found to have hemianopia with preservation of macular vision. The pathologic observations raised the question of the cortical localization of the macula. The author admits that the center is probably located in the occipital pole near the top of the calcarine fissure and that the innervation of the macula is bilateral, as suggested by Wilbrand. As the left optic radiation in this case was completely destroyed, bilateral innervation of the macula is the only way in which the retention of macular vision can be explained.

BERENS, New York.

IS PITUITARY SECRETION CONCERNED IN THE INHERITANCE OF BODY-SIZE? R. CUMMING ROBB, Proc. Nat. Acad. Sc. **14**:394 (May) 1928.

A comparison was made of the weights of the pituitary bodies in giant (Flemish) and dwarf (Polish) male rabbits and in their F_1 hybrids. There is no characteristic difference in the weight of the pituitary body that may be correlated with the differences observed in growth rate. With increase in body weight, a progressive decrease in relative pituitary weight occurs. In the full-grown dwarf rabbit, accordingly, there is relatively twice the amount of pituitary substance observed in the adult Flemish giant rabbit.

COBB, Boston.

FAMILIAL NYSTAGMUS. J. SOMMER and SEGALLER-MIRON, Ann. d'ocul. **165**:196 (March) 1928.

In a study of cases of ocular nystagmus occurring in a great number of people in the same family, the following conclusions are reached: (1) Nystagmus is familial; (2) it is connected with amblyopia and is combined with other ocular malformations; (3) the hereditary type is different in different families; (4) nystagmus is independent of the condition of the ear; (5) it appears after birth, therefore paralleling the increasing function of the eye, and (6) it is independent of hereditary diseases or of hereditary involvement of the central nervous system.

BERENS, New York.

THE CAUSES OF CATATONIA IN DEMENTIA PRAECOX. PAUL DESMAS-MARSALET, J. de neurol. et de psychiat. **27**:549 (Oct.) 1927.

After a study of the postural reflexes, the author claims that, contrary to common belief, catatonia is not a hypertonic manifestation comparable to parkinsonism. Investigation of the graphic, clinical and pharmacologic characters of the postural reflexes led to the conclusion that catatonia has nothing in common with postural reflexes. Physiologically, catatonia seems to possess all the characteristics of a voluntary motor action and possibly represents a negativistic attitude toward certain factors such as the action of gravity.

FERRARO, New York.

CYSTIC TUMOR IN THE REGION OF THE HYPOPHYSIS. J. BOURGUET and DESVIGNES, Ann. d'ocul. **165**:421 (June) 1928.

Desvignes, an ophthalmologist, began to have failing vision when he was 44 years of age. He consulted other oculists who found bitemporal hemianopia; roentgenograms of the skull showed marked enlargement of the sella turcica. Dr. Bourguet, who reports the case with Dr. Desvignes, operated and removed an embryonic cyst from one of the pharyngeal pouches. The cyst had developed in front of the gland, which it had displaced backward. Operation was not resorted to until fifteen treatments had been given with the roentgen ray.

BERENS, New York.

Society Transactions

PHILADELPHIA PSYCHIATRIC SOCIETY

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EVERETT S. BARR, M.D., *President, in the Chair*

INSANITY AND THE LAW WITH SPECIAL REFERENCE TO TESTAMENTARY CAPACITY. DR. LEROY M. A. MAEDER.

Insanity may be defined as a disease or disorder of the mind which effects a definite and more or less prolonged deviation of a person's mental processes, feelings, thoughts, ideas, judgment and behavior from what has been usually recognized as normal and customary for that person, in such measure that society and the law recognize that it is unjust to hold him to the same degree of responsibility in general as it does normal persons. Insanity is therefore a mental illness which incapacitates a man for the social relations of life in such a degree that the law takes cognizance of his condition and its effect on his social relations, and accordingly provides different criteria and rules of law by which to judge him and establish his liability or guilt in certain situations.

Insanity is not a medical term, but strictly a medicolegal term. As such it is composed of two elements, a medical element and a social or legal element.

The medical element of insanity is "mental illness," "mental disease," "mental disorder" or "mental sickness," and may be described as that general medical condition of the patient or symptom complex, of which the mental symptoms are an important or predominant part.

The first real point at issue in any proceedings at law in which the issue of insanity is raised is the fact of the existence or nonexistence of disturbed mental processes and behavior due to disease. Misbehavior due to disease will be considered as an excuse or defense, but an act or misbehavior not the direct offspring and result of mental disease or disorder will not be invalidated or excused.

The existence of mental disease is a fact to be determined in a scientific way. Its establishment rests on the testimony of physicians who have examined the man, although the final decision as to the existence of mental disease, or the mental element of insanity, rests with the court or jury. In frank cases, in which there is no controversy, the opinion of the examining and testifying physicians has the practical effect of decision. In borderline cases, when physicians may honestly differ in opinions as to the existence or nonexistence of mental illness, the court or jury makes a decision only after hearing much medical testimony pro and con.

The existence of the medical element once being established, the law immediately launches into a consideration of the legal or social element, which deals with the practical, social and legal aspects of the person's diseased condition. The law has recognized the general principle that a man who is so mentally diseased as to be morally and intellectually irresponsible should not be held legally capable, responsible or accountable. In pronounced mental disease or defect, the simple fact of obvious mental disorder in itself avails as an excuse or defense. In such cases there is the medical element of pronounced mental illness and the legal element, which is that the illness is of such nature and effect that it makes it impossible for the person to have the mental element or state of mind necessary for the performance of civil acts or for liability in criminal acts. The man may be said to be insane as respects all his legal and social relations. This statement, however, applies only to cases of this advanced type.

In mental disease of lesser degree, not obvious to all the world, the law holds that the mentally sick person can be conceived as exercising a reasonable judgment

in regard to particular acts and subjects, and holds that the mere fact of mental illness, without other pathologic conditions, does not exonerate him from civil or criminal responsibility, nor does it disable him to bind himself by a legally effective act such as a contract. The law, therefore, clearly recognizes the principle that there may be derangement of the mind or insanity in regard to particular subjects and yet capacity to act on other subjects.

The facts of mental illness and legal effect must coincide at the particular moment in question to constitute insanity.

A contract is an agreement enforceable at law. It comes into being through an offer and an acceptance of that offer, through a so-called "meeting of minds."

The parties to the contract must be capable and competent. To have the mental capacity requisite to make a valid bargain it is only necessary that a person have sufficient reason to understand the nature and effect of his act. He need not have average mental capacity. Mere mental illness, weakness of mind or even defect bordering on imbecility is not in itself necessarily sufficient to make an agreement voidable.

A person of unsound mind may contract a valid marriage, if he sufficiently understands what a marriage means and also the rights and the obligations following from it as regards the married couple themselves, their family and their property.

In torts, where a person inflicts a civil injury and damage on another through a legally wrongful act, not amounting to a criminal act, insanity is no excuse. The liability of a mentally ill person in torts for the payment of compensation is well settled in American law. The compensation is not by way of penalty, but as a satisfaction for the damage done to the one who has been injured in body or estate by him.

The American law of the liability of an insane person for his torts rests on several grounds. The rule has been invoked that where one of two innocent persons must bear a loss, he must bear it whose act caused it. It is further said that public policy requires this rule, in order that relatives may have sufficient motive and inducement to care for the mentally ill and deprive him of opportunities for inflicting injuries on others, and in order that normal tort-feasors may not simulate or pretend insanity to defend their wrongful acts which have caused injury to the body or estate of another.

A will may be defined as an instrument or disposition of property made by a competent testator in the form prescribed by law, to take effect after his decease, such instrument or disposition being revocable during his life.

A will is a legal document. It has the important effect of disposing of one's property after death. It therefore deserves serious consideration and every care in drawing up. As a legal document, it should be drawn up by a lawyer in order to insure a disposition of the property in accordance with the testator's wishes. However, in situations in which a will is most desirable but in which circumstances and lack of time make it impossible to call in a lawyer to draw up the will, the physician may be in a position to be of signal service to the testator and his family by having a knowledge of wills sufficient to enable him to assist the dying person in this capacity.

It is always well to have at least two, and preferably, three, subscribing and attesting witnesses to a will; the law of wills of the state in which the land lies governs this and it may vary in different states. To pass land situated in New Jersey, New York, North Carolina and Ohio, two subscribing and attesting witnesses are required; in Maine, South Carolina, Connecticut, Georgia, Massachusetts, New Hampshire and Vermont, three such witnesses to a will are called for. Each witness should sign his name under a statement at the end of the will to the effect that the witnesses have signed their names and witnessed the signature of the testator at his express direction and in his presence and in the presence of each other.

Any person, and especially a physician who signs his name to a will as a witness, should in all fairness and honesty to himself and to the testator be convinced before signing as a witness that the testator has a sound disposing mind,

for in acting as a subscribing witness the person or physician in effect declares it to be his belief that the person making the will has testamentary capacity. The physician, being ordinarily in a position to know the mental condition and capacity of his patient, really impliedly states that the testator has a sound disposing mind, memory and understanding.

Although the attending physician acquires his information regarding the patient's mental capacity while attending the testator in a professional capacity, the physician is allowed to testify in court when the capacity of the testator is in question, because the testator in asking the physician to act as a subscribing witness to his will by implication calls on him to bear testimony, if necessary, to all facts affecting the validity of the will. This in effect amounts to a waiver of the right accorded a patient by law to have his necessary communications to his attending physician considered as privileged and confidential.

To constitute a sound disposing mind the testator must, at the moment of making his last will, be able to understand that he is making a will, that is, the significance and effect of his testamentary act in effecting a disposition of his property after death; he must be able to comprehend, without prompting, the kind and extent of his property and the disposition to be made of it by the testament, and must be able to appreciate the persons who are the natural objects of his bounty and affection. To constitute a sound and disposing mind it is not necessary that the mind should be unbroken, unimpaired, unshattered by disease, or that the testator should be in full possession of his reasoning faculties. Less capacity is needed to make a will than is usually required for the transaction of ordinary business. Old age, sickness, distress or debility of body neither prove or raise a presumption of incapacity; nor will inability to transact business, physical weakness or peculiar beliefs and opinions.

It requires less business judgment to make a will than to make a gift. In the case of the pronounced mentally ill, there is no difficulty in determining that such a person is not capable of disposing of his property by will. But between the extremes of mental illness and a man of sound memory and understanding there is every shade of intellect and mental capacity. At what precise point or moment testamentary capacity ceases to exist it is almost impossible to state generally. The law must adapt itself to the practical needs of the greatest number; it must make allowance for individual variations, and it must therefore consider each case separately and on its own merits. Each case as to incapacity is, to a great extent, to be tested by its own facts and circumstances.

The right to make a will, the power of disposing of property in anticipation of death, is one of the most valuable of rights incidental to the ownership of property. It is conceded to the owner of property in every civilized country. It is obvious that it would work a severe hardship on the mentally ill to deny them this power simply on the basis of mental sickness per se.

RESPONSIBILITY IN THE LAW. DR. D. J. MCCARTHY.

To the understanding, scientific mind, the natural law is something definite and concrete with penalties for its violation. It is inexorable and takes no consideration whatever of ignorance or limited understanding or reduced responsibility.

The mundane law, on the other hand, the law of human beings, is something far different. It is concerned more with the convenience and comfort of the many than with the survival of the fittest. It prates much of justice, but is more concerned with individual rights than with the greatest good for the greatest number. The law, therefore, is the crystallization of customs into rules of conduct. All completely satisfactory laws are the embodiment of governmental authority and of rules of conduct, which the practice of the people has shown to be most satisfactory for the majority of the people governed.

In more recent times, the general tendency has been for a small paranoid minority to decide in advance what is the best thing for the whole people, and by hook or crook or unholy pressure to put such laws into effect for the chosen representatives of the people. A law like the prohibition law, so instituted, is

not enforceable. This is equally true of thousands of other laws, for the simple reason that they are not the crystallization of the thoughts and practices and customs of at least a working majority of the people.

Responsibility is a very practical thing. It is not esoteric or hypothetic, but something that from the beginning had to be defined as a workable proposition. Among primitive peoples, to kill a member of a neighboring tribe who had offended was not a crime. To kill a member of one's own clan might be a just act, but if the person killed happened to be the only spearmaker or boatmaker of the tribe, it became not only a personal matter but one that affected the interests of the whole clan and called for action. It would never to do permit valuable men of the clan to be so wiped out. And so the tribe in conclave appointed one of its own members — at first the chief, and later the priest, and later the wise man — who formulated the customs into precedents and law.

The wise man in the small clan or tribe knew each individual member and his characteristics. In dispensing justice he tinctured it with mercy for those of weak mental power or those of fiery temper. Those who were placed in positions of authority in the tribe were held to greater responsibility than the rank and file, and those afflicted with a God-sent insanity were held to no responsibility whatever; thus the doctrine of partial and complete responsibility gradually crept into the law.

Responsibility, therefore, was the reaction of the individual to the laws and customs of the tribe. Irresponsibility was a reaction away from this, in violation of these laws and customs.

In the development of civilization, the stress and strain on the brain power in clans, that had grown into hundreds of thousands and even of millions, became so great that the brain, which in simpler times was able to adjust itself to all the customs, rules and laws, found itself completely unable either to understand or to meet the new conditions.

One would, therefore, find a series of levels of intelligence which reacted to the new complex series of laws in various ways. At the top are found what might be called the superintelligence, the person of average or better than average brains, with power to stand exceptional training and high education, who often formulated the law and who frequently held himself, when necessary, above it. Next came the mass of people of average mind, who became the basis of custom and law because they were the producers and owners of property, and were most affected by it. Below this level was a great mass of people who were negligent of and negative to the law, but who through training and discipline adjusted themselves to it, because they were either benefited by it or dependent on the groups above them for their means of livelihood. This group was the group of potential revolution, who rebelled after submitting to the laws of the upper groups long after they ceased being accustomed to their well-being and comfort, mental or physical.

Finally, there was the lowest level who found the whole complex tissue of civilization entirely too much for their limited intelligence. They could not understand the law, its purposes and its aims, nor had they the brains to adjust themselves to its rules of conduct. Unable to support themselves in decency and comfort, they reacted as the primitive does, disregarding all law, taking what they needed when they saw it, killing their enemies and righting all wrongs by violence.

Practically all criminal law is concerned with this last group. Practically all psychologic investigation into crime concerns itself with an analysis of the mental caliber and the underlying reasons for criminal conduct.

The criminal mind is not anything definite or different from any other type of mind. If a mind, not adjustable to modern, complex civilization as lived in cities, be kept in relatively simple surroundings and treated sympathetically by the members of its clan or village, little difficulty is found with it, nor does it come into conflict with the law.

It is not at all surprising that a mind of limited intelligence, brought up from early childhood in slum surroundings where the atmosphere is one of resentment and rebellion against all law, should form the nucleus of the criminal class. A child of average mind brought up in such surroundings is apt to take the line of least resistance, and to drift into a quasi-criminal group. The child of full brain power realizes or may realize, apart from ethics and ideals, that honesty is the best policy and follows this pathway to the upper levels.

The law holds all men equally responsible, and unless insanity of the most advanced grade be shown, there is no excuse for crime. In civil matters a certain latitude is shown.

In dealing with people of relatively normal brains, one may assume that irrespective of their early environment or their education, in mass example, the total community must have a definite influence in molding the attitude toward what is right and what is wrong for those in immediate contact with them, for those in the neighborhood group and for the community at large. The public school in its effect is not purely ethical or religiously ethical, but a school where the child is taught to feel the effect of the human mass about him; of its customs and forms, and of what it approves and highly disapproves, and what will result in punishment.

There is a certain type of mind spoiled not in the making, but in the training, which knows only one way of getting what it wants, and that is to go after it directly irrespective of laws or regulations, mass feeling or what not. How soon this type of mind commits crime depends largely on how much resistance it has to the mass feeling, how much education and religion has done to reinforce this mass feeling and how much the fear of punishment has reached the sphere of feeling of the individual man. It is not a rule that the moron is the first to react at an early stage in this disregard of external pressure. Often it is the highest type of mind that feels he is above the mass, that his cleverness entitles him to the property and low life of others if he can "get away" with it.

Disease processes cause a variation in responsibility. The best example of this is the epileptic person. Certainly, if there is any one group of cases in the entire field of medicine in which a doctrine of diminished responsibility should obtain, it would be in this particular group.

In the alcoholic group, the law permits practically this doctrine of diminished responsibility by reducing the degree of the crime of murder, if it be shown that the act was committed in a condition of intoxication. The logic here is that a man intoxicated, who quarrels with a friend and strikes a blow killing him, is relieved, by his condition, of malice and premeditation.

One assumes that every human being is responsible for his acts unless it is demonstrated that there is such a defect of reason as to offer reasonable excuse. One should consider all the factors in the case. One should know first of all the ancestors of the subject with their thoughts and ideas, and the ideals which surrounded his early childhood. One should know not only the amount of brains they transmitted to the child, but also the thought atmosphere with which they surrounded him in his early developmental period, the physical and environmental atmosphere of the child up to and through adolescence, the physical diseases and the toxic factors in the blood stream through this period, the kind and quality of education — more particularly the general mental atmosphere of the educational periods — and above all the honesty of the sexual life more particularly during the whole adolescent period. The stress and strain of early adult life, new family atmosphere, the attitude of husband and wife toward the fundamentals of life, the honesty of purpose, freedom from hypocrisy, the methods of thought — all become of great importance in determining what the thoughts behind the offense were, how much the person himself or herself had to do with these thoughts and how much the early history, education, etc., had to do with a possible control. In dealing with deeds of violence, not only the religious training, but above all the racial factor, becomes of prominence.

The fault of the modern jury system in this regard was not originally a fault but a virtue. A man was to be tried by a jury of his peers, that is, his equals.

It was a neighborhood affair in a country district where twelve men good and true, who sat in judgment of the crime, had known the stock from which the accused came, his training and childhood environment, his religious training or lack of it, his quality of brain and his temper, and, knowing all these things, treated him fairly and with justice. But the man who is tried today by a jury is tried by men who know nothing of him, and any attempt to bring to their attention the things they should know is blocked by the rules of evidence which grew up around that old-fashioned jury, and which have not been changed to meet modern conditions and modern psychiatry. In minor offenses the municipal courts, in dealing more particularly with childhood, have made a sincere effort by their psychiatric clinics, social service and special investigators to hark back to the original system to get this neighborhood information of the victim's peers and to present it to the judge, not after the case has been tried as in New York, but before, in order that a real understanding of all those conditions that limit responsibility may be had in the investigation of the case during the trial and its final disposal.

The law, as it now stands, is the most practical solution of this business of full and limited responsibility. If the mental condition could be tried separately before the trial for the criminal offense, and if a wide latitude of evidence, as in the municipal courts, of the environment of the crime and early life of the individual were admitted, the degree of responsibility could be determined apart from the guilt in reference to the crime.

THE SENTENCE AND ITS EFFECT ON THE CRIMINAL. PAUL N. SCHAEFFER, President Judge, Twenty-Third Judicial District, Reading, Pa.

The idea of the protection of society, not a desire for vengeance, must guide the court in imposing sentence on an offender. It is an individual problem in each case. The first duty of the court must be to decide whether the protection of society requires the separation of the offender from society or whether the freedom of the offender, with or without supervision, is compatible with the common weal. If the decision is that the offender must receive custodial care, the court, or other agency, must select the institution which should receive him; but at the time of sentence no duration of imprisonment should be fixed, for that, judged from the sole basis of the protection of society, cannot be foretold; it must be dependent on the offender's reaction to corrective discipline. The determination of when the prisoner should be again accorded freedom should be entrusted to a parole board composed of experts in social science and psychiatry. Similarly, conditional freedom on probation should be for an indefinite period to be terminated by the social stabilization of the individual person. Much can be learned by the lawmakers and the law officers from the analogous problem of the medical practitioner in the field of epidemic disease.

DISCUSSION

DR. F. X. DERCUM: We have listened to three splendid papers. I have enjoyed all of them and also Dr. Burr's remarks. The difference of opinions is not as great as it appears on the surface, but I fully concur with Dr. Burr that we have a right to be protected against assault, against highway robbery and against the kidnaping of children and other misdemeanors. Certainly the remarkable and unprecedented prevalence of crimes of all kinds at the present day imperatively demands a sane and effective action on our part. It is important, I think, that one should recognize the one outstanding fact, namely, the biologic inferiority of the average criminal. A highly developed moral sense seems to have made its appearance only late in the history of man's evolution. In many criminals of today the moral sense appears to be entirely absent. Their development seems to have ceased at the level below that of even the caveman or the man of the stone age. Among the many criminals that I have had occasion to examine, I have never had one say to me "I am sorry." The main desire always was to evade the consequence of the crime. Regarding the more serious crimes, such as murder, I am informed that the practice in Germany before the war — what

it has been since the war I do not know — was to restrict the trial purely to the determination of the facts of the crime, i. e., the question of insanity was not allowed to be raised at all. If the jury brought in a verdict of guilty against the prisoner, the counsel for the defense had the right, if he so chose, to arise and to address the court, stating that he believed his client to be insane. Such a statement having been made, it was the simple function of the judge to order the prisoner's commitment to an institution for the insane with the order that the physicians in charge of the hospital should make a report on the case of the prisoner at the end of about three months. This was done with the object of avoiding the often discreditable appearance presented on the witness stand by opposing medical experts. I believe that if such a plan were adopted by ourselves it would likewise achieve this most desirable end.

The proposition of instituting a plan of probation is a method that does not appeal to me, but, as far as the lesser crimes and misdemeanors are concerned, it may perhaps prove effective and may lead to the redemption or saving of an occasional prisoner. The fact must not be lost sight of, however, that the great mass of persons convicted of crime are persons who are biologically inferior, of whom we speak habitually as defectives and delinquents, and the outstanding right and duty of the law to protect the community against such persons must be unfailingly upheld.

JUDGE PAUL N. SCHAEFFER: Dr. Dercum, I think I have failed to make myself understood. I would want to restrict probation for the first offenders, but to serious felons one cannot apply probation.

I should be very glad to receive some advice from you. Do you believe it possible to set up a means by which the men now in the penitentiary could be tested or studied; that there would be any possibility of determining whether or not they could be set free without further danger to others?

DR. F. X. DERCUM: I think in some cases you might be successful, but I think such cases are comparatively rare.

DR. J. H. LLOYD: The subject of testamentary capacity has been referred to by Dr. Maeder. This has always seemed to me the most difficult subject in the medical jurisprudence of insanity. In criminal cases the man is before us; one can examine him, test him and fight over him. But in the case of the contested will the testator is dead, and the case must be carried on by evidence. It is almost impossible to make a scientific study.

Take the subject of lucid intervals. That is an old-time legal phrase, not a medical one. What does it mean? It dates back to the time when lunacy was thought to be caused by the moon. A lunatic was called after luna, the moon; consequently, he had lucid intervals at the changes of the moon. It is extraordinary how this subject has been treated by the judges. Some have been capable of seeing lucid intervals under any circumstances. There is the case of Deaver vs. Weaver in which a man was said to be insane every other day, and it was decided by the court that he had signed the contract on one of his sane days. That was a Pennsylvania case. Another was the case of a man said to have had insane delusions for three days before signing his will. He committed suicide the day after he had signed the will, and the court decided that he had had a lucid interval on the day he signed.

I refer to these cases to show how some ideas promulgated by the courts are impossible of acceptance by psychiatrists. I think some judges have been dissatisfied with them too, for some of the courts have tried to put aside this old definition.

The same thing can be said about delusions. To what extent can delusions impair the validity of a will? The subject of delusion started in the case of Hatfield, who was defended by Erskine in a speech which is classic. Hatfield was an old soldier who had fought in the Napoleonic wars and received severe wounds in battle, one in the head. He became unbalanced in mind and delusional. One night he shot at the king and was tried for high treason. He was defended by Erskine on the ground that he had delusions, and that these delusions impaired his moral and legal responsibility. It was the first attempt to establish delusions

as a defense. The next step was to take it into civil cases and to establish the rule that insane delusions impaired testamentary capacity. This led to the decision by Lord Brougham, the eminent English chancellor, that a man with insane delusions is an insane man. It was remarkable because it was the first real scientific decision on the subject.

Now what does the law require? To make a will a man must be of sound and disposing mind. Brougham said that he is not of sound mind if he has insane delusions. This decision was a scientific one. Would any alienist say that a man whose life was influenced or ruled by delusions is going to be free from them just at the moment when he is making his will? Is it given to any judge or jury to say so? But Lord Brougham's decision was not acceptable to the judges; it was too radical.

Some time later, there was the case of Banks vs. Goodfellow, the case of a lunatic who had been confined in an asylum and who was still insane when discharged. He was epileptic and unable to transact business, yet his will was declared valid because it was not shown that his delusions had influenced the testator in making his will. I believe that is the way the law stands in England and in America today. Unless the delusions can be considered a direct influence on the making of the will, the will is valid.

The law says that a man to have testamentary capacity must be of sound and disposing mind and memory. But that is defining in a circle; it is simply giving a different name for a thing; saying that a man has a sound and disposing mind and memory is simply saying that the man is not insane. It leaves the whole subject of insanity open. A delusional lunatic or imbecile may make a will. A delirious patient or an alcoholic paranoid patient may make a will.

Some years ago, in New Jersey, there was the case of a man, aged 80, who had been a chronic inebriate for many years. He was broken down with alcohol and was senile. He had entertained the delusion that his only son and only child was not his son, but the son of his wife by another man. He made a will disinheriting that son entirely and the law supported it. Why? You will have to read the judge's decision to understand it, if you can do so then. He tried to make a distinction between a delusion and entertaining a rumor. How can you do it? Yet the court supported it. If you will examine the law reports you will find that all sorts of decisions have been arrived at. A certified lunatic has been allowed to make his will. One of the chancellors allowed a man in a lunatic asylum to make his will. A patient dying in the delirium of pneumonia has been allowed to make his will. The only way to decide such cases is by the methods of science; that is, by making a diagnosis based not on presumptions and old legal definitions, but on the symptoms in each individual case.

DR. SAMUEL LEOPOLD: In regard to Dr. Burr's supposed pessimism, which I think is really a modified optimism, I believe that society needs to be protected, but I believe that the individual criminal should be considered in the handling of any case as well as society. I think that the individual person should be reconstructed. I do not agree with Dr. Burr and Dr. Dercum that we should look on this as entirely hopeless. Of course, there are a certain number who are undoubtedly incapable. Work is still being done on the subject of cancer, and although the problem has not been solved conditions have been improved somewhat. I think the same is true in these cases.

Little has been said about the preventive side of this problem. When one considers that there has been little attempt to handle even the younger offenders in an adequately scientific fashion, that does not prove that the younger group is entirely hopeless. I do not like to look at the offender as constitutionally inferior. I like to look at him as a person who has failed to become socialized. In some instances that may be due to glandular defect or to marked hereditary taint, and according to the statements of one of the Child Guidance Clinics in the West, at least 50 per cent could be traced to the parents.

DR. LEROY M. A. MAEDER: Testamentary capacity is not only an interesting, but also a complicated subject. For that reason I have limited myself

to a presentation of the pronouncements of the Supreme Court of Pennsylvania on testamentary capacity and sound disposing mind and to its statements regarding the effects of delusions and defect of memory, understanding and knowledge on this capacity.

DR. D. J. MCCARTHY: What I tried to point out was exactly what Judge Schaeffer brought up in his discussion, namely, that if one is going to judge the possible potential criminality of a series of first offenders in early life, one must readjust those persons. If one can take a series of these first offenders appearing in the Childrens' courts and make conditions of life simpler for them, where the community will know them, I think there will be a large percentage of readjustments. If one takes a man out of prison and drops him in a neighborhood where no one knows him, that man is pretty sure to be a second offender within a year. If, however, one takes him to some district where life is simple and everybody comes to know him, I think there is a good deal of hope. If one takes even a mental defective or a moron or one of a "bad-egg crowd" and puts him in the right sort of surroundings and treats him decently he will undoubtedly be a reconstructed moron.

JUDGE PAUL N. SCHAEFFER: I want to apologize here, although I did not know that I would get a second chance. If I gave anybody the impression that I advocate probation and parole in all cases I certainly failed in the reading of this paper. I do believe in probation and parole, and have three times as many men on parole as behind the bars. But that is not as many as in Massachusetts. I get weekly reports on these men. Not only do I get weekly reports, but I have a man who goes out and checks up. They cannot give reports that are not true for more than two or three weeks. I do not think anything of this kind can be supervised from the office alone.

The Huntingdon Reformatory has no parole system worthy of the name. They have two boys in jail today who are mentally defective. One of them shot a policeman, but fortunately did not kill him. That is a serious problem, and these fellows should be sent away for life; yet under the law they cannot be given more than seven years, or rather from three and one-half to seven years, and about 90 per cent of the chances are that they will get out at the end of three and one-half years. It is, however, not the fault of the court, but of the system.

Another thing I wish to mention is regarding what Dr. Leopold said about juveniles. In Philadelphia we have a home where we can keep twenty-five, and the success of that home has been remarkable. It has been running for about twenty years, and, although statistics are not available, I believe that about 90 per cent of the children have kept out of trouble. There is not a bar or lock in the whole place; any one can run away if he wants to. The children go to public schools. It is just each boy's home.

Regular Meeting, Nov. 9, 1928

E. S. BARR, M.D., *President, in the Chair*

THE CHILD OF DISSATISFIED PARENTS. DR. FREDERICK H. ALLEN.

The relationships and attitudes of different members of a family group are factors which strongly influence the making of a child's personality and behavior. The parents' attitude toward a child is determined largely by the degree of satisfaction and contentment they have been able to achieve in their own adult life. The child of contented parents rarely develops into a problem or later into a psychoneurotic or psychotic person. Discontented parents tend to overidentify themselves with the child and to relive their own lives in the life of the child. This type of relationship quickly entangles the child in the emotional difficulties of the parent and frequently leads to difficult behavior or an unhealthy personality, because it places a definite barrier in the growing-up process of the child.

An illustrative case showing the operation of parental discontent in the creation of a behavior problem is that of a boy, aged 10, who was extremely jealous of a younger brother and showed a striking lack of confidence in his adjustments at school. The treatment was directed, not to the behavior of the boy but to creating a better adjustment of the adults toward each other; if effected this would remove the unwholesome emphasis they were placing on that behavior which incited a fear that the boy was developing traits similar to those which have been so disabling in their own lives.

Some phases of modern life add to the element of discontent: crowded living quarters, drab life of the mother, particularly in the poorer economic groups, absence of the father from home, particularly in the commuting centers, and the element of fatigue. Extra home activities for parent and child have helped to lessen these factors and to broaden the relationships of child and mother and to reduce irritation. The wholesome use of these facilities, not as an escape from the drabness of home life, but as a chance to develop worth while interests and relationships, is one means of counteracting parental discontent which bears such an important causal relation to the problem child.

SOCIAL FACTORS IN PSYCHIATRIC CASES. DR. EARL D. BOND.

Last June, I was so much interested in certain sociologic facts brought forward by Dr. Plant, that since that time I have been noticing psychiatric cases in point. Some of these cases I am going to present without much discussion and with the hope that they will make a setting for Dr. Plant's paper.

Case 1 provides a contrast. Here all the economic and social factors are exactly right, and the accident of disease is the thing that puts everything askew. This man, aged 45, has worked for an old manufacturing concern for twenty-five years, holding more and more responsible positions. He had an adequate income to support a happy family. An encephalitic process hit him, lost his position and upset the whole economic foundation of this family.

Case 1 showed acquired disease as the overwhelmingly important factor. Case 2 showed congenital defect. A boy, born with hypospadias which operation when he was 11 years of age failed to cure, showed ideas of inferiority which led to depression and finally a withdrawal into unreality—into dementia praecox.

There are also cases which result from an upsetting blow from the economic side. A man worked his way up from errand boy to proprietor of a grocery store in the mine regions. The last coal strike knocked out his business, and he began to be depressed and confused.

A bit more complicated is the case of a saloon-keeper whose place was padlocked. He had been a steady drinker at his work. The closing of his place led to melancholia.

Another man was a great drinker. The passing of the prohibition act made him bitter, and an honest effort to increase the amount he drank in proportion to his indignation resulted in an alcoholic collapse.

Several alcoholic patients, especially from the better circles, have been faced with a dilemma on leaving the hospital. On one side there are overwhelmingly medical reasons for not drinking; on the other, an overwhelmingly social pressure from all their friends to drink.

Similarly, there are boys with neurosis whose environment—riches—offer them so much without work that healthy incentive is lacking; even the old fashioned chores are gone.

Whether this social pressure is given a weighty enough place in the counsels of many physicians is doubtful. In a case at the Pennsylvania Hospital, a young man with an aptitude for athletics was found to have a heart murmur. His family physician kept him out of athletics, forgetting the sociologic facts; he had plenty of money and naturally turned to a dissipated crowd. He has been absorbing liquor, good and bad, by the gallon for twenty years, and his weak heart is standing up grandly under the strain.

One often meets the sociologic element for the first time when discharge from a sanitarium is being considered. A most striking instance is at hand. A woman,

whose ten years of involutional melancholia were spent in a sanitarium, recovered fully and was ready to go home. Her husband had married her best friend, and the two were living together in her former home and using all the furniture she had gathered.

A final consideration is of the sociologic situation about many postencephalitic children. In general, the overactivity in them called for certain adaptations in their homes which could not be made. How could one expect a mother to care for a household, including younger babies, and find time to amuse and occupy a restless and irritating young patient who did not look sick? How could a mother be expected to take the impersonal attitude which the treatment of the disease demands? How could the well-being of the sick child be promoted at the expense of that of other, especially younger, children? How could a father, coming home tired at night, fit into such a difficult situation? In one case, in which a father did try to do everything for the boy, he lost his job. And how can our graded schools care for such unusual problems as postencephalitic patients present?

One boy, by the way, had the encephalitic-like symptom of sleeping by day and being restless all night. He slept with a drunken father who beat him as often as he could remember to do it, and who would not reverse the sleep rhythm with such a sociologic fact in bed with him?

The problem with the children seems to be in miniature that with many psychiatric patients, and with some unusual people who are not psychiatric. For the treatment of the disease, certain things are needed — flexibilities, understandings, long distance plans — which laws and occupations and customs do not allow one to have in the patients' homes.

THE EFFECT OF POPULATION CONCENTRATION ON THE CHILD'S SENSE OF SECURITY. DR. JAMES S. PLANT.

The adjustments to reality and authority are stated to be mental habits — rather rigidly established early in life and coloring all later mental reactions. A distinction is made between "reasonable" and "unreasonable" authority — the latter referring to those forces so foreign to a rational formulation of life as to render adequate "compensation" impossible. Reasonable authority leads to the problems of submission and rebellion; unreasonable to stability and anxiety states. In a large metropolitan area the child finds the excessive population — movement and restlessness — as of the nature of unreasonable authority. Despite the fundamental character of this threat one finds oneself helpless to deal with it as its motivation so far transcends individual control.

DISCUSSION

DR. E. A. STRECKER: I feel that I am not able to discuss these papers without some preparation. They constitute a contribution of definite value, and offhand I am not able to give any valuable discussion.

DR. E. S. BARR: Most of us feel that the papers presented at this meeting and the points which Dr. Bond has touched on are so important that they stimulate thought rather than immediate discussion.

DR. PEARSON: I was much interested in Dr. Plant's opinion that by the time children have reached school age they have acquired certain rather definite behavior patterns. I have been making a study of preschool children, and it seems to me that certain personality reactions and types of behavior are rather definitely formed by the end of the preschool period. When one considers the effect of parental attitudes on the child, attitudes that were formed in the parents before the conception of the child, attitudes that the parents have formed toward the child due to their desire or lack of desire for the pregnancy — due to the mother's health during the pregnancy, the discomfort, etc., she might have suffered at the birth, the illnesses of the child during the first year of life — in short the influence which the interplay of reactions between the parents themselves and

between the parents and the child must exert on the child's developing personality during the first two or three years of life, it makes one feel that the real future of the child's personality and his behavior depends much more on this period of his life than it does on the purely theoretical conceptions of the inheritance of behavior and personality traits, and is even more vital than the influences which affect the child after school has been started. In my study the children have carried into the school situation the personality traits that have developed during their preschool life.

The situation is rather serious when one has to deal with a child whose parents are dead or whose parents are antisocial. Although the child may be put in a good foster home, he must have considerable feeling about the loss or actions of his own parents, and this feeling may be contributed to by chance remarks of the foster parents who usually know something of the child's background. I know one girl, aged 15, who developed a bitter hatred toward her mother because the mother placed her in a foster home when she was 3 weeks of age. I would like to ask Dr. Plant if he has found any method by which children can be given a more wholesome feeling about these real facts in their life history. Would it seem advisable to develop a friendly contact between the adult and the adolescent child, and through that contact attempt to give the child some insight into why the parents, if antisocial, had to behave toward the child as they did? Adolescent children frequently work out their own difficulties with their parents by adopting the tolerant, understanding attitude of an adult to a child. Would such an attitude help children of antisocial parents in their adjustment? If some such technic cannot be developed to treat these problems in the adolescent child, it seems inevitable that one must wait until late adolescence when the child can undergo a thorough analysis and through this work out this difficult problem.

DR. F. H. LEAVITT: At the Child's Guidance Clinic which I was conducting at the Children's Hospital before the All Philadelphia Child's Guidance Clinic came into being, I was impressed by the number of children brought to the clinic by foster parents — people who have never had children of their own, but who have wanted them, and have taken the means of satisfying their desire by adopting a child. The majority of these children were obtained through an agency — in a great many instances they were illegitimate, in other instances they were children from families where there have been disarrangements which made it necessary to dispose of an infant. These foster parents have provided the best kind of environment, and yet these children have been brought in with mental defects, conduct disorders and an inability to adapt themselves to a normal environment.

I present this fact as being somewhat along the lines of Dr. Burr's remarks. It seems to be the congenital make-up of the child and not the environment that is at fault as these children have been taken from a bad environment to a good environment, yet their abnormalities are markedly manifested.

DR. J. S. PLANT: I feel, as Dr. Burr does, that Dickens was a great man — so great that he serves to prove not only Dr. Burr's point, but also my own. It is true that we picture him as a great humorist, but he was a man of the bitterest and most cynical criticism. The paper was not about people who are defective or social misfits. One sees successful business men, so-called "leaders," lacking completely in what I would call the worth while things of life — showing underneath a fundamental anxiety and lack of feeling at ease that arises out of some childhood situation. A man going through life biting and snapping at other people is not a "normal" man; that is, from the point of view of happiness he is not a "normal" man.

Dr. Burr's second point is is it right to take these children away from their homes? I am much in doubt about the value of taking them from their homes and their parents to give them money and position. I think perhaps that Dr. Leavitt, in supporting Dr. Burr, is not so definitely supporting him as telling us that it is interesting that these foster children — despite the fact that they have position, "nice homes," etc. — are not really healthy, happy souls just because one

has taken from them precisely these most basic needs. I do not know. I have not seen these children. I simply make this suggestion.

I have used one old-fashioned concept "faith." May I use one other? There is the old statement about "The sins of the father being visited upon the children even to the third and fourth generation." We say that now in this way "the conduct-patterns of the parents appear in their children — generation after generation." I do not see why we are not willing to face the fact that many matters of parental conduct definitely stamp themselves on the children — and that psychiatrists are helpless in the matter of therapy in regard to them. That is not nice to admit, but it is so.

What should one do with the "bad parents" of children? Many of us have tried to twist these parents' lives into some beautiful picture. It is possible with those who come out of this "bad parent" end of the chart — through the school and foster home — to give the veneer of stability to the child. Of course, there are foster-parents who give the child quite a bit of security. Then you also see the other side. Take for instance, the case of a boy who was failing in school, a typical bully; they kept failing him and eating away at his sense of security; adjustment after adjustment was tried. Now he is moving toward the upper end of the chart — the bully reactions giving way to those of more furtive anxiety. It is my general feeling that toward the top of the chart there are the more malignant conditions; and, toward the bottom, the more benign.

Dr. Burr has asked me if the bully reaction in this particular boy was not largely bluff. I feel that it was not. I have known only two or three children who have really accepted failure. They usually meet it by going somewhere else to say "I rule here." They go back to their mothers or to younger children and lord it over them (the "spoiled" child or the "bully"). These tend to be certain reactions which you do not get in the anxiety and uncertainty types. That child feels that if every one else beats him down he can still rule the mother or some smaller child.

Dr. Burr has also asked me to define "Faith" again.

I cannot define it. There are many facts absolutely foreign to what you or I think life should be. You cannot make death rational to me, or 7 o'clock in the morning. There are certain definite factors in life that are absolutely unreasonable in the sense that we would never have put them in were we constructing the affair. No "compensation" can make up for them. In talking to the parents about "faith," I use a term which is itself unreasonable — "The feeling that everything will come out all right." Still I insist that what I am trying to get at is something which I cannot define — I think there are some things one cannot do.

DR. E. D. BOND: I bought a book today called "America Comes of Age," and I expect to find in it some description of such communities as Essex County. As I listened to Dr. Plant I kept thinking of the America that was a pioneer community — where it was supposed to be a good thing for families to move up through the different stages of society. Now in crowded communities one sees some moves of families which are detrimental instead of helpful. It probably means that pioneer ideas do not work in a neighborhood which has 10,000,000 people in it.

BLOOD PRESSURE IN MANIC-DEPRESSIVE PSYCHOSES IN ITS POSSIBLE RELATION TO THE PROGNOSIS. DR. ALFRED GORDON.

Psychiatry, after a long period of purely descriptive existence, entered recently a domain of interpretative speculations. For the latter it had to join hands with psychology. Some serious observers still view psychiatry only from the standpoint of anatomy or physiology. Experience in the domain or psychiatric problems convinces one that orientation in one special direction will not solve the complex phenomena in psychiatry. As a branch of medicine, the latter must utilize all the resources of knowledge including abnormalities in feeling, in thinking, in behavior and in the reactions to external stimuli. The present contribution makes an attempt to determine a possible relationship between a physical phenomenon in the cardiovascular sphere and certain psychotic manifestations.

After a detailed analysis of the variations in the manic phase, the depressive phase and in control cases, Gordon shows that a relationship between an alteration of blood pressure exists in manic-depressive psychosis. Moreover, it presents some tangible means to foresee, to anticipate, to foretell modifications or more or less oscillations, or terminations of a given phase of the psychosis.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 15, 1928

LOYAL DAVIS, M.D., *President, in the Chair*

FAMILIAL CEREBRAL PALSY IN CHILDREN. DR. HARRY A. PASKIND.

These three patients were from the clinic at Northwestern University Medical School. They were brothers, aged 9, 8 and 6 years, respectively, the first and successively born in a family of four children. The mother had no miscarriages. The fourth child, aged 2 years, was apparently normal. All three presented a clinical condition: cerebral palsy as manifested by spastic paraplegia (in one child there was also spastic palsy of the right arm). All three were idiots and had epileptic seizures. Such a syndrome corresponds well with that of Little's disease.

Although the clinical pictures were striking, there was a noteworthy lack of etiologic factors. The parents were not consanguineous. At marriage, both were 25 years of age; all three children were born within four years after marriage. The father was delicate as a baby and did not walk until the age of 3. From the tenth to the fifteenth year he was subject to fainting spells, which occurred at irregular intervals, mostly when he was hungry or in a poorly ventilated place. During these spells he experienced an increasing sense of physical weakness and would faint if he did not get into the open air. Twice he became unconscious; when consciousness returned he was well oriented. The father's educational and industrial histories were good. For nine years he successfully conducted a hardware store of his own. He has always been able to provide for his family and now owns his home. The mother's personal history was without significance, except that she was shy, sensitive and somewhat irritable. Previous to marriage, she was a piano teacher. A paternal aunt, at 20, had a "nervous breakdown," apparently an attack of manic-depressive depression lasting for two months. It is curious to note that a maternal uncle at 5½ months was able to walk while holding on to furniture and was able to say "mama." There was no evidence of alcoholism, tuberculosis or syphilis in the parents. Both had a negative Wassermann reaction of the blood. Pregnancy in each case was normal. Injury and uterine diseases cannot be of etiologic importance here because it is extremely unlikely that all three would be afflicted in a similar way at corresponding periods of life. Labor was in no case difficult; with the oldest patient it lasted for eight hours, with the next three and three-quarters hours, with the third one and three-quarters hours. Forceps were not used and there were no dry labors. Postnatal asphyxia and convulsions were absent. The patients all took to the breast readily and were well nourished. It was noted very early that all three were inactive, rigid and hard to dress. Sitting and crawling were late in all three. The oldest began to walk at 7½, the next at 5, and the youngest at 3 years; the gait in all had always been of a modified "scissors" type with genu varus and pes valgus. None of the patients had yet talked. Seizures began in the oldest at 2, in the second at 7, in the third at 5 years, and had continued to recur since the onset. All three children were untidy. Evidence of injurious postnatal influences, such as meningitis, encephalitis, cerebral neoplasm, etc., was not found. This would be expected since it is unlikely that all three would be attacked by such factors.

There is little to account for such a pronounced condition in the three brothers. The neuropathic taint in the family is not greater than that found in the families

of thousands of normal persons, certainly not great enough to account for such a devastating process in so many siblings. Consanguinity, parental syphilis, tuberculosis, alcoholism, plumbism, etc., were absent. Pregnancies and labors were normal in each case. Infantile deleterious influences were not found. It would seem that there must be some mysterious injury to the germ, a blastophthoria, or perhaps these cases illustrate the theory of "germinal enmity," a condition in which the male and female reproductive elements are in themselves normal, but their fusion produces an abnormal product.

DELAYED TRAUMATIC SERRATUS PARALYSIS. DR. JOHN D. ELLIS (by invitation).

Isolated traumatic paralysis of the serratus magnus muscle is much more frequent in men than in women; it is also more common in the active period of life and on the right side. For convenience of description the nerve may be divided into three parts: (1) The point of origin from the cervical roots of the plexus. Isolated damage to this part of the nerve is practically impossible. (2) The three roots of the nerve in their course through the scalenus medius muscle; this is the most vulnerable portion. (3) The portion below the scalene muscle. This part is protected from all but penetrating wounds or severe injuries to the wall of the chest.

The causes of injury to the second portion of the nerve may be divided into: (1) prolonged pressure on the nerve as by carrying heavy weights on the shoulder; (2) compression of the nerve from contraction of the scalene muscles, and (3) blows or force applied to the base of the neck over the scalene muscle, particularly when this muscle is strongly contracted.

The symptoms of serratus magnus palsy are characteristic. The classic inability to raise the arm above the horizontal is often compensated for by the action of other muscles, at least in part. Before concluding that a palsy of the serratus magnus is traumatic, disease of the nerve by distant pyogenic infection, syphilis and compression of the nerve roots by osteo-arthritic deposits must be excluded.

Delayed traumatic paralysis has been described by several writers. The following cases are instructive from this point of view.

CASE 1.—A. W., aged 43, sustained a slight cerebral concussion and multiple trivial lacerations in an automobile accident on Sept. 8, 1927; his right shoulder was struck against a post. On the next day, there was a bruise with hardness of the underlying muscles over an area about 4 inches (10.16 cm.) in diameter about the middle of the upper border of the right trapezius muscle. Nothing of note was found on examination of the arms in motion. Four days later, he complained of inability to use the right arm properly and on examination at this time there were signs of serratus magnus palsy, and the arm could not be raised above the horizontal. After three months of treatment, the paralysis was still apparently complete and the serratus still failed to react to faradism, yet the arm could be raised to about an angle of 45 degrees above the horizontal. This improvement was due, therefore, to compensatory development of uninjured muscles. Complete recovery occurred in this case in ten months.

CASE 2.—A man, aged 30, fell so that the region of the middle of the upper border of the trapezius muscle struck against a timber. When examined immediately after the accident, there were no signs of serratus magnus palsy. About two weeks later, the typical deformity began to appear. Within a further period of two weeks, this became more marked than in case 1, but in six weeks no deformity was obvious in the resting position of the arm.

DISCUSSION

DR. LEWIS J. POLLOCK: I was much interested in Dr. Ellis' analysis of the function of the serratus muscle. Dr. Davis will remember that we constructed an anatomic model, attempting to study the function of the muscles which abduct the arm, and were fairly well in agreement with the conclusions of Dr. Stookey. The third part of the movement of abduction of the arm was concerned in part

with the deltoid muscle. The serratus not only produces a movement of the whole shoulder girdle, but also makes it possible for the supraspinatus and clavicular head of the pectoralis major to pull the head of the humerus into the glenoid cavity to produce an adequate fulcrum and leverage.

RABIES IN CHILDHOOD. DR. MAURICE L. BLATT (by invitation).

The inadequacy of words to picture neuromuscular disturbances in childhood interested me sufficiently to put a moving picture apparatus into the County Hospital, where a large number of children with neuromuscular disturbances present themselves. Among the group of cases we have had an opportunity to photograph during the last eight or ten years is this series of cases of rabies. Rabies is not a very important subject from the standpoint of neuropsychiatric practice. It is only important because it is a unique disease in its manifestations, because so large a motor area is involved clinically, and because of the type of muscular activity, which is peculiar. It has to be differentiated from hysteria, manic excitement and tetanus. Those three diseases almost cover the field of differential diagnosis. None of these presents a picture such as one sees in rabies. The coordination of muscular activity is quite different from what one sees in convulsive tic. It is different from what is seen in meningitis of the tuberculous type, in which it is common to see the finer type of tremor. Tetanus presents a problem in differential diagnosis. The facial muscular contractures, the rigidity of the neck and the history and incubation period aid in differentiation.

The diagnosis of rabies is made in the admitting room. The patients come in with a history of a dog bite and an onset that is suggestive. In our experience the course has been short. We shall have more cases, because rabies is on the increase all over the world, and particularly in this district. I have a map showing the relative status from 1921 to 1926. The entire southern group of states is violently involved. The southern part of Illinois was reached through Ohio, and by 1929, the northwestern part of this state will be touched. Following the war, the epidemic was pronounced in Europe. In England and Ireland there had been no cases of rabies for many years until brought in by airplane from the continent. There have, I believe, been a few cases in England, but none in Ireland. Australia has always been free, but whether this will continue cannot be said. Ireland requires a six months' quarantine for all animals brought into the country. The history of the cases I am showing in moving pictures is as follows:

CASE 1.—The incubation period was two months. E. S., aged 9 years, was bitten by a dog on the tip of the nose and the left leg. He was treated locally with mercurochrome-220 soluble. The prodromal symptoms were drowsiness for forty-eight hours and delirium for twenty-four hours. An attempt to drink water resulted in a convulsion. There was bloody vomitus and expectoration. The temperature rose to 104.5 F. Short periods of delirium alternated with periods of perfect lucidity. Death occurred twenty hours after entrance. Treatment consisted in morphine, ⅛ and ⅙ grain (0.008 and 0.01 Gm.); encephalitis serum, 20 cc., and tryparsamide, 1 Gm.

CASE 2.—The incubation period was three weeks. F. B., aged 2½, was bitten by a dog below the left eye. He was ill four and one-half days—drowsy, refused food and slept. These symptoms were followed by sudden twitching, convulsions and twisting of the body. He was excitable, with a staring expression and rigidity of the neck. No pathologic changes were found in the heart, lungs or abdomen. Treatment consisted in 1/32 grain of morphine (0.002 Gm.).

CASE 3.—Incubation period was about three weeks. L. F., colored, aged 6, who was admitted on July 14, 1928, had been bitten by a dog below the left eye. The illness was of thirty-six hours' duration. The temperature was 105 F.; vomiting, convulsions and foaming at the mouth were present. The eyes reacted slightly to light; the neck was rigid, and involuntary movements of all extremities were noted. A laboratory report showed 29,550 white blood cells, with 80 per

cent polymorphonuclear cells and 20 per cent lymphocytes. Death occurred in eight hours. Treatment consisted in rabies serum; morphine, 1/12 grain (0.005 Gm.), and 10 cc. of 25 per cent magnesium sulphate. The nurse reported that the child was sensitive to external stimuli and the movements of other persons.

CASE 4.—C. S., colored, aged 5, was admitted on Nov. 13, and died on Nov. 14, 1927. He complained of abdominal pain, nausea and vomiting of three days' duration. There had been convulsions, fear and fever, with no loss of conciousness, of one day's duration. He had been bitten, seventeen days prior to admission, on the forehead, bridge of the nose, arm and leg. He was treated locally at a hospital by hypodermic injection (antitetanic serum, possibly). He was restless and had the appearance of great fright, but was able to swallow a little water. Laboratory examination showed blood sugar, 88; Wassermann reaction, negative; culture of spinal fluid, negative. The temperature was 103 F.; the pulse rate, 106, and the respiration rate, 36. The Pandy test was negative. Treatment consisted in chloral hydrate and calcium bromide, 20 grains (1.3 Gm.) of each. Rabies vaccine was administered intramuscularly twice.

DISCUSSION

SIR CHARLES BALLANCE: I spent three weeks in Pasteur's laboratory at about the time his treatment for hydrophobia was published. I remember him very well. He was intensely enthusiastic. What struck me was his extraordinary kindness to the animals he had in the laboratory, and to the patients he saw. I saw several patients who had come from Russia with terrible wolf bites. He made a vast number of dogs immune to rabies. I have been much interested in this presentation. We have stopped these epidemics in England by means which could hardly be used in this country, and the chief menace now is the rich women who come in from the continent with their dogs in their muffs. That ought to be considered as a criminal offense. Whenever I see a case of hydrophobia, I feel that every dog in the world should be destroyed. We had at one time an epidemic among the deer in Richmond Park, near London, and they had to be destroyed. A bite from a deer would be as bad as that from a wolf. I believe that in the cases shown, the patients all had bites on the face, and of course that is much more dangerous than in other places—one or two were on the nose or eyelids, I think. What struck me was that the speaker said nothing about the Pasteur treatment. I do not know whether it is used here or not. In England the antivivisectionists have prevented the establishment of a Pasteur institute, so the material comes from Paris.

DR. SAMUEL J. HOFFMAN: These pictures of Dr. Blatt's undoubtedly speak for themselves. The anxious expression of these patients, the marked irritability and the marked salivation I have found to be characteristic. With reference to the incubation period, Kozwaloff reported 212 cases in which two years was the highest incubation period. There were three cases with a one year period, eight with 220 days of incubation—the majority of cases incubating for from twenty to forty days. Koch thought that the virus lies dormant in the central nervous system for some time and such factors as trauma, fatigue, alcoholism and acute infectious diseases may precipitate the attack.

At the County Hospital, we have followed the work of Semple. We use a fixed virus taken from the brains of rabbits and placed in sterile saline containing 0.5 per cent phenol. The virus is killed by phenol and incubated at 37 C. for twenty-four hours. This is then injected into an experimental animal, and if the latter does not develop rabies the virus is put on the market. I have treated sixty patients and have had no systemic reactions and no treatment paralysis. In cases of severe dog bites of the face we use nitric acid and phenol as a cautery, then follow this with twenty-one injections. Fourteen injections are used in ordinary bites of the body. Little is known about the active treatment for rabies. We had an interesting case awhile ago. A rabid dog bit a cow, and the cow died of rabies. The nine children that drank the milk of this rabid animal came in for the Semple treatment. The father of these children refused treatment so we

shall watch him eagerly as a control. Some investigators have reported cures from active rabies by the use of various drugs, but it is believed that these were cases of lyssophobia.

SIR CHARLES BALLANCE: An amusing incident occurred while we were doing some various experimental work at Brown Institute. A gardener came in with a letter asking that he receive Pasteur treatment because he had been bitten. We found that he was working for the vice president of the antivivisection society. The employer was informed that we would not treat the man unless he removed his name from the roll of the antivivisection society. This he did immediately.

DR. MAURICE L. BLATT: In reply to Sir Charles Ballance concerning the Pasteur treatment, these were neglected cases. All cases of dog bite are in the same class as gunshot wounds. They are reported to the health department and treatment is compulsory. The last patient shown was treated locally and neglected. These children are from poor environments. If the incident is not reported the patient is not treated, and all of these came to us practically without treatment and after too long a period for treatment to do any good. The law is stringent, but these cases have occurred just the same. We apologize for them, but we can do nothing more. There are a number of institutions in which treatment is given free; the only difficulty is the lack of police power which would allow one to take all such patients in for treatment. Education will do a great deal, particularly in the groups of colored people who are not well educated, and in a group from southern and eastern Europe who are hard to reach. It will take one more generation to accomplish anything with them. We have the same difficulty with antitoxin. The way to combat the epidemic is by muzzling dogs, by getting rid of strays and by inoculation with serum. Of 17,000 or 18,000 cases, less than 0.1 per cent of patients developed the disease and that was in the middle of the epidemic; of 4,000 reached and not inoculated 0.9 per cent developed the disease.

INTERMITTENT MUSCULAR SPASMS RESEMBLING JACKSONIAN EPILEPSY, COMPLICATING RECURRENT EPIDEMIC ENCEPHALITIS. DR. T. B. THROCKMORTON.

This article will be published in full in a later issue of the Archives.

DISCUSSION

DR. PETER BASSOE: I want to express my congratulations to Dr. Throckmorton for reporting this interesting case which brings out so many important and perhaps not so unusual symptoms of encephalitis; for instance, the occurrence of repeated acute attacks, each one being more or less typical of encephalitis. One symptom is extremely unusual—hemianopia. As to convulsions, many of you heard Professor Wimmer's address in Cincinnati in 1926, in which he covered the ground fully, and a good deal of discussion has arisen as to whether the epileptic manifestations are due to injury to the cortex or whether they may be produced from striatal lesions.

DR. B. LEMCHEN: In this case I should like to ask if a Lange test had been made. We have found changes in the spinal fluid in a good many instances; they have not the curve of paresis, but we do find changes in some tubes. I have seen them even a year after the onset of the disease.

SODOKU TREATMENT OF PARETIC PATIENTS: PRELIMINARY REPORT ON SEVENTY-TWO CASES. DR. A. S. HERSHFIELD (by invitation).

Seventy-two paretic patients, thirty-seven women and thirty-five men, were inoculated with rat-bite fever, after the method of H. C. Solomon, of Boston, during August, 1927. The injections consisted of 0.2 cc. of blood from the heart of a guinea-pig, subcutaneously, into the upper and inner aspect of the thigh. The incubation period varied from eight to fifteen days. The temperature varied

from 102 to 106 F. The fever was permitted to run an average of thirty days before the disease was terminated by arsphenamine.

After one year's observation the results were as follows: Of the total group, 50 per cent were more or less physically improved; 20 per cent were from slightly to markedly improved mentally; 10 per cent were paroled and doing well in civil life; 60 per cent showed no change mentally, and 10 per cent at first showed improvement and then became worse. There had been ten deaths, of which only two could be attributed directly to the infection.

The physical and mental improvement of the patients was encouraging, and rat-bite fever should be given consideration either as a primary treatment or as an adjunct.

DISCUSSION

DR. B. LEMCHEN: I agree with Dr. Hershfield that the temperature is not the factor in the improvement one sometimes observes in paretic patients treated with foreign proteins. During the epidemic of influenza I treated a number of patients suffering from paresis. Some of them had a temperature as high as from 108 to 110 F. for as many as eight days. A good many of them recovered from the influenza, but the mental condition was not improved.

DR. A. S. HERSHFIELD: The primary purpose of the treatment was to find out how it compared in efficacy with malaria and typhoid. At Elgin, in the absence of material from a rat or guinea-pig, a patient was sick with rat-bite fever and we inoculated ten women with blood from this patient. Not one developed the disease, from which we concluded that rat-bite fever is not transmissible from man to man. We always examine the blood before inoculating the patients. The rat-bite organism should be shown in the blood of the guinea-pig before inoculation is done. The more recent the onset of paresis, no matter what the age of the patient, the better are the results of the treatment. Paresis of long duration gave the highest percentage of failures. We had total failure in more than 60 per cent of the patients inoculated, which is higher than with malaria or the nonspecific protein forms of treatment.

CORRECTION

"ELECTRONYSTAGMOGRAPHY: A GRAPHIC STUDY OF THE ACTION CURRENTS IN NYSTAGMUS," BY DR. I. LEON MEYERS, LOS ANGELES

In Dr. Meyers' article in the April issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, the graphs in figure 3, on page 907, now labeled *B*, *C* and *CC*, should be labeled *D*, *E* and *EE*, and those labeled *D*, *E* and *EE* should read *B*, *C* and *CC*. On page 914, 6 *E* in the first line should read 6 *D*, and 6 *C* in the second line should read 6 *B*.

Book Reviews

ANATOMIE COMPARÉE DU CERVEAU. By R. ANTHONY. Price, 70 francs. Pp. 355. Paris: Gaston Doin, 1928.

This is a most interesting contribution, dealing largely with the gross anatomy of the brain. Most students of cerebral comparative anatomy have devoted themselves to a study of the tracts and nuclei; that is, the comparative anatomy of brains cut into microscopic sections. Professor Anthony takes the brain as a whole, discusses its weight, its general form, its flexures and its axis.

In the introduction there is a review of the structure and function of the elementary nerve cell, and a description of the division of the nervous system into the cerebrospinal and sympathetic systems. The embryologic development of the cerebrospinal system is described. The general form of the brain is discussed in chapter 1: the structures visible from the superior aspect of the brain of mammals are telencephalon, metencephalon, and myelencephalon, with rarely the corpora quadrigemina and epiphysis. In man only the telencephalon is visible from the top. On the inferior aspect all five lobes are visible. The arrangement in mammals is due to two conditions: the expansion of the dorsal regions of the metencephalon and telencephalon, and the development of flexures. These are enumerated as:

(1) Primary flexure (cephalic flexure) at the level of the mesencephalon with its concavity directed ventrally.

(2) Cervical (nuchal) flexure, at the level of the myelencephalon is the second flexure to appear. Its concavity is directed ventrally. This flexure is most accentuated in the upright animals, corresponding to the position of the head in relation to the spinal column.

(3) Pontile flexure, at the level of the metencephalon, the concavity of which is directed dorsally; this counteracts the other two.

(4) Telencephalic flexure, at the level of the corpus striatum. The concavity is directed ventrally. This is the last one to appear, corresponding to the development of the telencephalon, and giving to it a more and more spherical form.

The brains of most mammals have a straight base line, continuous with the axis of the body, and a vertical cerebrocerebellar cleft. To this general arrangement there are two exceptions:

(1) In man and monkeys, the accentuation of the cephalic flexure and the growth of the telencephalon result in the latter more or less covering the cerebellum. The cerebrocerebellar cleft becomes more and more horizontal, so that seen from above only the cerebrum is visible in man.

(2) In cetaceans accentuation of the pontile flexure gives to the myelencephalon a dorsal concavity.

The weight of the brain is the principal theme of this first part of the book. The absolute weight of the brain is first considered. In tables it is shown that decreasing brain weight corresponds only in a general way to the decreasing body weight. The larger animals are high in the list, with heavy brains. Primates, especially man, are out of order. Moreover, comparison of the weights of different individuals in the same species show similar irregularities. Hence the weight of the body is seen to exert a direct influence on the weight of the brain, but it cannot be the only influence. When the relative weight of the brain and body is tabulated, the ratio favors small animals; so attempts to reach a more accurate method of classification are described. For example, Brandt showed the relation between the size of the brain and the body surface, rather than weight. Small animals have greater surface in proportion to weight. Since greater surface calls for greater chance of response to environment, smaller animals have correspondingly greater brains.

Dubois reached the problem from a more empiric approach. He took two animals of the same genus but differing as widely as possible in weight, and sought to find some power (r) which would correct the discrepancy arising from the difference in size already noted. For example:

	Brain Weight	Body Weight
Cat	30 Gr.	3,000 Gr.
Lion	250	135,000

$$\frac{30}{250} = \frac{(3{,}000)r}{(135{,}000)}$$

By many comparisons of different species "r" was found to average 0.56; it is named the "exponent of relation."

Dubois then tries to determine a "coefficient of corticalization" (K), from the formula: brain weight = K x Body Weight x 0.56. This gives a possibility of figuring the "degree of corticalization." Many tables are given with observations on a great variety of animals.

From the table on page 49 it is seen that mammals arranged according to "K" fall into an order much in accordance with the commonly accepted idea of their intelligence. Man is at the top and near the bottom are the hippopotamus, the marsupials, the rodents and the insectivores. Discrepancies, however, still appear. It is dangerous to try to give a numerical value to intellectual faculties. For example, the cetaceans appear too high in the table. This is due perhaps to excessive myelination of the axons. The elephant also stands high in the list, possibly for the same reason.

There are several interesting results obtainable by application of Dubois' formula. A comparison of sexes shows that the average weight of the male brain is 1,360 Gm. and the average weight of the female brain is 1,220 Gm. and the difference is 140 Gm.; that is to say male: female = 100: 90. The body weights of male and female are then taken into consideration, and it is found that in proportion to body weight the female brain is heavier than the male brain. This agrees with the earlier observations on relative weights of larger and smaller individuals of one genus. Using Dubois' formula, the K (coefficient of corticalization) gives the male 2.73, and the female 2.74. A corresponding ratio of male and female can be found among lower species.

The Growth of the Brain is studied by considering the relation during various stages in the growth of the animal of the following factors: (1) the absolute weight of the brain, (2) the relative weight of the brain, (3) adult brain weight — brain weight at a given stage of development, (4) "L'indice de valeur cérébrale."

The last is developed from an application of Dubois' formula. It is the ratio of brain weight, at a given period of development, to a calculated weight of the adult brain, if the adult were reduced to the size of the immature individual at the chosen period of development. This gives what one might consider as the degree of corticalization at a given age in the growth of the individual. An analysis of the table of these "valeurs cérébrales" shows significant results. There is a steady growth during intra-uterine life and a slowing down toward the end of that period. The index attains a value of 1 at about 1 year of age. Then there is a steady growth reaching a maximum at 7 years of age. From 7 to 30 years there is a steady decrease, but the index is above 1 until age 40. From 40 to 90 there is a steady decrease. Women show less decrease than men. It is interesting to note that the maximum is at about 7 years of age, which "corresponds exactly to the period of life during which the mind is forming and in which the brain makes the greatest effort to acquire the gifts that it will utilize during the rest of life." An analysis of the absolute weight shows rapid growth between the sixth month of intra-uterine life and the seventh year. From 7 to 20 there is slow growth. From the ages of 20 to 60 the weight is stationary with perhaps a tendency to decrease toward the end of this period. After 60 there is a distinct decrease, which may be as much as 300 Gm.

Chapter 2 considers the telencephalon as a whole, taking up first the form of endocranial casts and their changes during development. Each lobe of the brain is then discussed, and finally the form of the ventricles. Chapter 3 is concerned with the rhinencephalon; the olfactory apparatus of macrosomatics is first dis-

cussed, leading up to a description of these regions in primates. This is largely gross description with many good diagrammatic illustrations.

Chapter 4 continues a thorough study of the fissures and configurations of the neopallium. The development of the operculum is interestingly described from the comparative point of view, also the calcarine fissure. The embryologic development of the fissures in man and some mammals is then considered. Finally the cyto-architecture is reviewed, and the tracts are briefly described. Lastly, in chapter 5, there is a discussion of the basal ganglions, with a good summary of the recent theories of function. The illustrations, as in other parts of the book, are helpful to the student because they are diagrammatic. References are fairly numerous, and an index of authors cited is appended.

THE PROBLEM CHILD AT HOME. By MARY BUELL SAYLES. Price, $1.50. Pp. 329. New York: The Commonwealth Fund Division of Publications.

The basis for misconduct in the cases coming into a child guidance clinic is to be found largely in the emotional life of the child. The growing child has definite emotional needs; one of the most important of these is a sense of security, of which the first condition is love from the parents. Balancing the need for security, but not antagonistic to it, is opportunity for growth and freedom, to be acquired in easy stages, for although without such opportunity the child may eventually break away from parental ties, yet he will always carry over immaturities into adult life. In the development of personality, example is better than precept, but even example may fail if too deliberately emphasized. If parental ideals are low, the child may either follow or reject them, but conflict is often involved in the rejection of a parent model. On the other hand, when the ideals or accomplishments are high, the danger of discouragement to the child is to be avoided. As an aid to normal emotional development the companionship of a parent without immaturities and without rigidity is almost essential. Between these emotional needs of the child and the parents' own search for emotional satisfaction, there is often a conflict. Excessive devotion and service in early years, while a pleasure to the parent, cultivates helplessness and dependence. Inconsistent discipline is common in such cases. This exaggerated parental love is often found in a parent who has had a disappointed adult love life, perhaps due in turn to his relationship with his own parents. A demand for satisfaction of parental ideals, frequently along educational lines but also in standards of manners, morals and religious practice, may be the basis of conflict, a conflict which increases when the parents are of a dominating type. The factors back of these parental ideals and needs and the mechanisms by which they affect the emotional life of the child are discussed by the author. Other factors affecting the child are antagonisms and jealousies toward siblings, resting on real or fancied favoritism in the parents, a situation often seen in the eldest child, who, displaced by the advent of a second, is driven to attract attention by undesirable behavior. Mistaken ideas of child nature, of disciplinary methods, an emotional attitude toward sex behavior and a false conception of the influence of heredity may be the cause of conflicts between parent and child. Twelve illustrative cases from the records of child guidance clinics are given in detail in the second half of the book. This volume is to be particularly commended for its emphasis on the rôle of parental maladjustments in behavior problems. It is a work which will be of value to the physician and social worker as well as to the parent.

PECULIARITIES OF BEHAVIOR. By WILHELM STEKEL. Cloth, $8.50 per set. Vol. 1, pp. 315. Vol. 2, pp. 329. New York: Boni & Liveright, 1924.

Detailed studies of cases are presented showing those peculiarities of behavior known as impulsive acts. Kleptomania, pyromania, dipsomania and similar conditions are included. Stress is laid on the psychogenesis of such types of behavior, and the significance of various contributing factors is disclosed in the exposition of each case. As in the author's previous publications, *"Fortschritte der Sexualwissenschaft und Psychoanalyse,"* little if any theoretical interpretation of psychoanalysis is given. A single chapter only is devoted to the treatment of instinct,

affect and impulse. Stekel is not interested in nomenclature, although the science of psychology, and particularly that branch of it with which he deals, is so undeveloped that the first necessity of all sciences—an exact terminology—would seem imperative as a foundation for fruitful thinking. Mere categorical statements of the author's point of view—a point of view which differs only in minor aspects from that of Freud—are disappointing. The assumption of many disputed facts and principles will impress psychologists as a defect that may detract from the value of the studies themselves. Thus, a fundamental assumption of the author is that all instincts are reducible to those of hunger and love. The objective of these volumes, however, is primarily to describe analytically types of impulsive acts and to suggest their genesis rather than to arrive at any synthesis of psychologic principles; this objective is attained. There is little in the way of novelty of ideas. The cases include diseases that have been treated in a somewhat similar fashion by Freud and other psychiatrists. The claim of the translator that "the significance of these mental manifestations is revealed for the first time" cannot be maintained.

In general, the translation is excellent. One slip, however, demands correction. On page 324 of volume 2, the term "high school" is used to translate what actually must have been institutions of higher learning or professional schools. It is preposterous to suggest that psychoanalysis should be studied in high school; yet this might be supposed from the translation—an unfortunate recommendation since the present tendency is to diffuse the meager knowledge already attained rather than to attain further knowledge of the laws of mental functioning.

STAMMERING. By ISADOR H. CORIAT. Price, $2. Pp. 68. New York: Nervous and Mental Disease Publishing Company, 1928.

This book is number forty-seven of the "Nervous and Mental Disease Monograph Series." Its main thesis is that the oral-erotic components of stammering are so important that the term stammering should be dropped and the term "oral neurosis" be substituted. The older theories of stammering are discarded as "worthless" and "unscientific conjectures." In discussing treatment, Coriat says: "Heredity, neurotic disposition, auditory amnesia, cerebral congestion and wilful imitation have in the past been too largely incriminated as the cause of stammering. These explanations are purely superficial and have led to the minute elaboration of useless phonetic methods of treatment, which are merely a mechanistic surface plowing. The originators of these methods have not realized that it is futile to teach the stammerer how to talk, because under certain circumstances and definite situations the stammerer experiences no difficulty in speaking. In the phonetic methods of treating stammering, any benefit derived is only temporary, as most stammerers quickly relapse and go from one system of treatment to another. Such phonetic methods treat nothing but physical stammering: that is, the speech defect alone, while the complex character-traits of the stammerer, his infantile reactions, narcissism, resistances, the entire ego and libido development, are left untouched and unchanged." Such a statement carries conviction.

Elsewhere stammering is defined in these words: "it is a regression to the earliest level of childhood, an arrest of development at this stage, a form of gratification of the oral libido which continues as a post-natal adult gratification of nursing." This is a distinctly interesting theory, but the reader who wants the evidence on which this definition is based will have to look elsewhere. In the whole sixty-eight pages of the book, there are only about two pages that could be called data; even these observations do not agree with those of other investigators. The other sixty-six pages reiterate the author's views in an increasingly tedious way. The book is an ex cathedra presentation of analytic doctrine.

DIE BEHANDLUNG DER QUARTÄREN SYPHILIS MIT AKUTEN INFEKTIONEN. DR. BERTHOLD KIHN. Price, 22.50 marks. Munich: J. F. Bergmann, 1927.

One can do hardly more in attempting to review this work than call attention to its extraordinary completeness. Twenty-three of the 316 pages of text are given to a consideration of the specific treatment of paresis, all the rest being

devoted to a consideration of fever therapy in all imaginable aspects. The author considers the febrile method so much superior to the specific methods of treatment that he is able to dispense with a further discussion. One may criticize, however, the fact that he has apparently not taken into consideration, either through his own experience or through reading of the American literature, the effect of pentavalent arsenic such as tryparsamide. This is a defect in the consideration of the whole problem in the treatment of paresis.

On the other hand, when it comes to a consideration of the febrile methods, one can find no fault. It is a treatise that is thorough, and one can find a complete account of the situation as it existed at the time when the author wrote the work. He gives the history of the febrile methods, and includes in this discussion malaria, relapsing fever, and rat-bite fever, as well as the various chemical and protein methods of inducing fever. He reviews the anatomic changes that seem to follow therapy. The clinical and serologic results of treatment are adequately presented, including the reports in the literature and in his own experience.

A full bibliography is included. This work certainly contains everything that one needs to have concerning the status of febrile therapy up to 1927.

LA SYPHILIS MÉDULLAIRE. LORTAT-JACOB and POUMEAU-DELILLE. Price, 14 francs. Pp. 110. Paris: Masson & Cie, 1927.

The authors offer, in this short monograph, the more recent acquisitions in the field of syphilis of the medullary portion of the cerebrospinal system. The work is divided in a logical manner, beginning with a brief historical résumé, followed by a discussion of the pathologic anatomy of each form considered. This is treated in a fairly complete manner, but the remainder is written in the characteristically sketchy style of the French.

The chapter on pathogenesis, whether one agrees or not with the authors, is rather stimulating, especially the discussion of two types of vascular involvement which result in the production of acute myelitis: (1) that with which one is familiar, an obliterative arteritis with a resulting destruction of the surrounding tissues, and (2) a form which they term "vasoparalytic." The cord is said to show extremely dilated and markedly engorged intramedullary vessels with an edema of the adjacent tissues. They explain this dilatation of vessels by an inhibition of the sympathetic fibers.

Mention is made of Sicard's method of producing a polyneuritis for the control of excessive spasticity by giving frequent small doses of the arsphenamines. The treatment considered otherwise offers nothing new.

The bibliography is moderately complete.

COMMON PRINCIPLES IN PSYCHOLOGY AND PHYSIOLOGY. By JOHN T. MACCURDY. Price, $6. Pp. 301. New York: The Macmillan Company, 1928.

Psychology and physiology, sciences dealing with the same organism, have developed for the most part independently. This volume is an excellent attempt to provide a common foundation of general principles for both. It is evident that much profit will be derived from the synthesis made. It is a unique attempt to scrutinize thoroughly the foundation of two sciences concerned with closely allied fields of study that now ignore the challenge of their opposed principles.

Wood-Jones and Porteus in "The Matrix of the Mind" have recently called attention to the state of many psychologic textbooks, which give a brief inadequate treatment of the nervous system in the first chapters, and subsequently deal with sensation and attention, as if these mental functions did not have any relation to neurologic phenomena. Maccurdy's service lies in providing a basis for the study of the unified organism. He proposes that patterns be taken as the basic principle in the two sciences. He tries to synthesize all the facts and to harmonize the observations of the various schools of psychology. The presentation is an interesting study of fundamental theory in the tradition of the English critical school. Its publication at a time when there are psychoanalysis, behaviorism, and Gestalt psychology in addition to the traditional psychologies, is appropriate and valuable.

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